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mansoura medical students community

MANSOURA
MEDICAL

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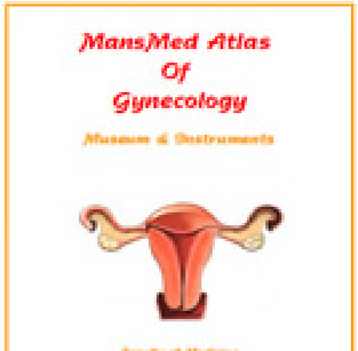
MansMed
Forums

Welcome to MansMed Forums

ATLAS


MansMed Atlas
Of
Gynecology
Museum & Instruments

falling A.P.A.R.T

Oral: Mention ٣ طرق for Aq. formation?
① Ultrafiltration
② Diffusion
③ Active secretion

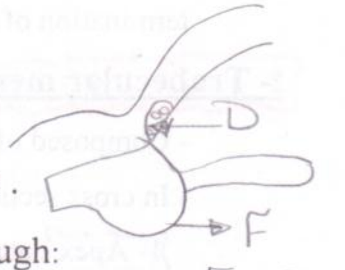
or طريقتين for Aq. formation?
① Passive process
② Active secretion

GLAUCOMA

Introduction

Aqueous Humor

It is a transparent fluid that fills the ant. & post. chambers .



F = D

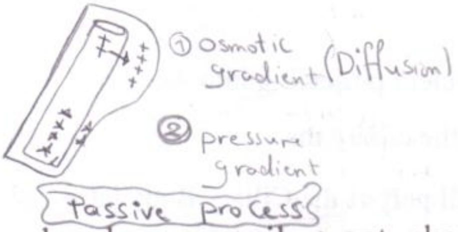
- بيدخل بطريقتين **Aq formation :** by the ciliary body processes through:

① Active secretion : carbonic anhydraze enzyme is needed

② Ultra - filtration

③ Diffusion

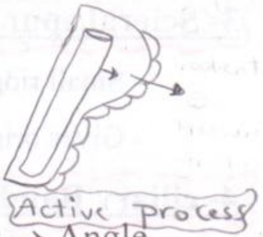
Passive



① osmotic gradient (Diffusion)

② pressure gradient

Passive process



Active process

- Energy
- B-receptors
- CA enzyme

- **Circulation :**

Ciliary body → posterior chamber → pupil → ant. chamber → Angle

- بيخرج بطريقتين **Aq. Drainage**

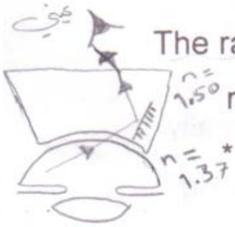
① Mainly (85%) : Through the angle

② Small part (15%) through the C.B & iris root (uveo – scleral outflow)

Anatomy of the Angle

Def : It is the recess that lies between the periphery of the cornea & the iris root .

- The angle of the ant. ch. cannot be examined except by **Gonioscopy** because



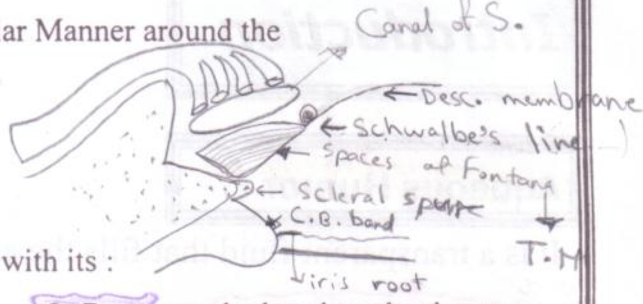
The rays coming from the angle undergo total internal reflection (TIR) at the cornea -air interface.

* To overcome this → a contact lens is placed on the cornea (Gonio-lens) to change the cornea - air interface into cornea - glass interface & so → No Total internal Ref.

The following structures can be seen by Gonioscopy : Written

1- Schwalbe's line :

It is a condensation of fibers in a circular Manner around the termination of Descemet membrane .



2- Trabecular meshwork

- Composed of parallel laminae
- In cross section, it appears triangular with its :

1- Apex : attached to the Sch. Line   2- Base: attached to the scleral spur.

3- Scleral spur :

- Small ridge of sclera projecting into Ac.
- Gives origin to the ciliary ms .

4- ciliary Band : small part of the Ciliary Body base

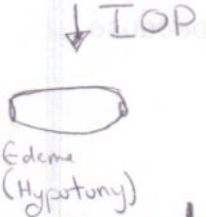
5- Root of iris

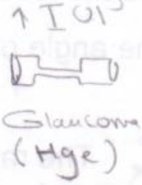
- ***The following structures are Not seen on Gonioscopy :***

1- Canal of Schlemm : Circular canal (endothelial - lined) inside the limbus

2- Aqueous veins : Drain the aq. from the canal of Schlemm to the episcleral veins .

Intra - ocular pressure





Normal IOP : - It is the pressure under which the eye function normally

The average IOP Ranges between 10-22 mm Hg

(with diurnal variation of about 2-3 mm Hg)

(The difference between the 2 eyes is usually < 4 mm hg)

Diurnal variation:

- Normally the IOP is highest in the morning & goes to minimum in the evening (the difference is about 2-3 mm Hg)

causes: ① Extra - ocular ms massage. (طول الليل عضلات العين مش شغالة وده بيقلل drainage بتاع Aq. ويبقى الصبح IOP أعلى ما يمكن ولما العضلات تشتغل طول اليوم بتساعد drainage فيقل IOP آخر اليوم)

② Venous stagnation during sleeping → ↓ aq. outflow.

③ Circadian rythm of steroids

Measurement of IOP :

(1) Digital method: *rough method (like indentation of a cyst)* → لما بتحسس على cyst لا تفرق منها غير الـ surface

عشان أحس الضغط مباشرة ← sclera.

المريض يعمل إيه → *While the patient is looking down without closing his eyes.* → to avoid Bill's Phenomenon

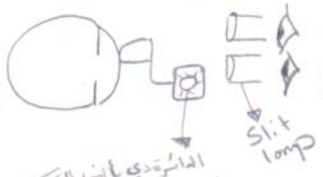
الدكتور يعمل إيه → *Pressing by one finger& receive the impulse by the other.*

(2) Tonometry

ⓘ Indentation Tonometry (Schiotz tonometer):

While the patient is supine , the tonometer is placed perpendicular to the cornea which measure the IOP by measuring the degree of corneal indentation produced by known weight placed over the cornea .

Disadvantage: affected by scleral rigidity



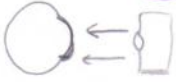
الدائرة دي بأقدر بيها التحكم في المسافة اللي هتضغط على العين لجوه

ⓘⓘ Applanation Tonometry(Goldman tonometer):

which measure the IOP by measuring the force needed to flatten a small area of the cornea (3.06 mm)

- With patient seated at the slit lamp, a cone(3.06 mm) is used To applanates an equal area of cornea.

Adv.: more accurate (less affected by the scleral rigidity) .



ⓘⓘⓘ Air puff tonometer: non-contact tonometer.

أهم مميزاته → less infection & manipulation.

Primary site of damage in glucoma is the (nerve fiber layer) as it passes through the optic disc

Glaucoma

لاستى
Triad

Definition : It is a ① pathological IOP leading to optic ② nerve damage (optic neuropathy) & visual ③ field defect.

N.B. : The important factor in glaucoma is not only the IOP, but also the perfusion pressure of the ocular vessels which depend on the difference between the blood pressure in the ocular vessels & the IOP.

So, (1) ↑ IOP +Op. n. damage + field defect → Glaucoma

(2) ↑IOP + Normal op. n. + normal field → Ocular Hypertension (مش محتاج مني غير مجرد follow up)

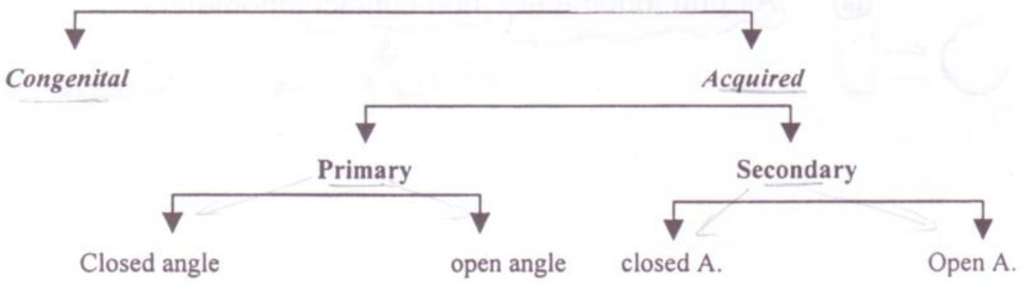
(3) Tn + Op. n. damage + field defect → low tension glaucoma.

المريض شايف أمامه ستارة سوداء

ضغط قليل صحيح بس كافي على المريض

Aqueous outflow facility = 4 times aqueous formation.

Classification



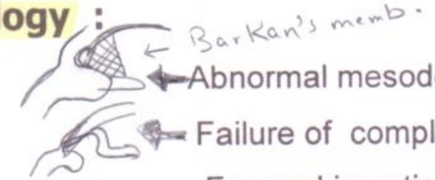
Congenital Glaucoma

Written (v.imp)

(1ry Buphthalmos)

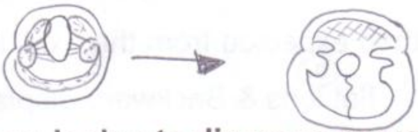
Definition : It is elevation of IOP due to congenital anomalies closing the angle

Etiology :



- Abnormal mesodermal membrane in the angle. (Barkan's membrane)
- Failure of complete separation of iris from the cornea
- Forward insertion of ciliary ms to the trabecular meshwork

- Absence of canal of Schlemm (result rather than a cause) كده ؟ → (Disuse atrophy)



N.B. ***2 ry Buphthalmos :*** **is due to diseases or syndromes as**

(i) Retinoblastoma , ROP , Neonatal iridocyclitis, Trauma.

(ii) Sturge- Weber syndrome (Ipsilat. hemangioma in face, meninges &choroid)

(iii) Congenital rubella , Lowe syndrome.

Incidence :

① Age : 80% of cases are presented before the age of 6 months.

② Sex: more in boys (Male: Female 2:1)

③ Side: commonly bilateral (75% of cases) → Congenital وراثي حاجة

④ Hereditary : commonly autosomal recessive

Clinical picture :

***symptoms** : given by the mother

(1) Early : lacrimation, photophobia, blepharospasm (الـ ٣ اللي ممكن الأم تعرفهم من الـ ٥

(due to irritation of the corneal nerves by the edema) .

(2) Late : large eye, Hazy cornea

شكوى الأم : عين ابني كبيرة

Oral v.imp

****Signs :** The eye will distend with ↑ IOP as the outer coat is still elastic

① Cornea : - Diameter ↑ - Curvature ↓

- Transparency ↓ (Haze) (أم جاية تشتكي على عين ابنها الكبيرة)

- May also show : Haab's stria (due to rupture of Descemet memb.) ↓ very thin in children

② Sclera : Bluish (Being thin ,showing the choroid)

③ A.C : deep

④ Pupil : large

⑤ Iris : tremulous

⑥ lens : flattened & displaced backward كأنها

⑦ Tension : high

⑧ Fundus : shows optic disc cupping or atrophy.

⑨ Gonioscopy : reveals the abnormalities of the angle . هانعرف سبب المشكلة

كبير theuritical ⑩ Refraction : Myopia (but less than expected from the axial length)

due to: ① flat cornea , ② flat lens & ③ Backward displacement of lens .

Diagnosis

depends on

early or late أعرف (1) Measurement of corneal diameter

cong يبقى (2) Gonioscopy :

glucoma أعرف إن كده (3) Measurement of IOP (under ketamine anesthesia)

المادة الوحيدة اللي بتحافظ على tone بتاع E.O.M

general anesthesia لأ
→ relax EOM → ↓IOP

Differential diagnosis :

from other causes of :

(1) Corneal enlargement : as Megalocornea , congenital myopia

(2) blue sclera

(3) Watering of the eye : congenital dacryocystitis

Complications :

1- Loss of vision : due to optic atrophy

2- ↓Of vision : due to : (i) complicated cataract, subluxation of the lens. Due to disturbed metabolism (↓ nutrition of lens due to stagnation of Aq.)

(ii) Nystagmus (in bilat. cases) & Amblyopia (in unilat. cases)

لازم ألحق الولد قبل 6 years

Oral v.imp

Signs : The eye will distend with ↑ IOP as the outer coat is still elastic

(1) Cornea : - Diameter ↑ - Curvature ↓

- Transparency ↓ (Haze) (أم حاجة [illegible] على عين ابني الكبيرة)
- May also show : Haab's stria (due to rupture of Descemet memb.) ↓ very thin in children

(2) Sclera : Bluish (Being thin ,showing the choroid)

(3) A.C : deep

(4) Pupil : large

(5) Iris : tremulous

(6) lens : flattened & displaced backward كأنها

(7) Tension : high

(8) Fundus : shows optic disc cupping or atrophy.

(9) Gonioscopy : reveals the abnormalities of the angle . هاتوريك سبب المشكلة

(10) Refraction : Myopia (but less than expected from the axial length) theuritical

due to: ① flat cornea , ② flat lens & ③ Backward displacement of lens .

Diagnosis

depends on

early or late أعرف (1) Measurement of corneal diameter

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[illegible] ألحق الولد قبل 6 years

Treatment : Oral v.imp

(I) Medical Treatment (Not alone)

***Indications:** 1-Preparation for surgery.

2-Adjuvant after surgery.

***For Examples:** (i) Beta-blockers.

(ii)Topical CAI. (Diamox) → ↓active formation
Process

(B)Surgical Treatment:

1-Early cases :with Corneal diameter less than 13 mm, (Canal of schlemm is still opened)

أي تعامل
مع طفل
(أ) عمرها
الطفيفة
قليلة

- ***If the cornea*** is clear ,do→ Goniotomy

If the cornea is Hazy ,do → Trabeculotomy .

(Canal of schlemm لسه مفتوحة)

*** Goniotomy** (cut in angle) → limbal incision to do operation in the opposite side

-Under visualization with a gonioscope ,

-A goniotomy knife is introduced from the limbus to cut the mesodermal memb.

Trabeculotomy :

① Limbal incision to the Schlemm's canal(بأدخل إلى حوصلات الأعلى نقطتين)

② a fine metal probe is passed into the canal .(canal of Schlemm في needle تدخيل)

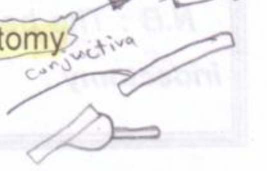
③ The probe is swept into the anterior chamber.

Principle : to expose the Schlemm canal directly to the aq. in the ant. chamber.
↓
No trabecular meshwork
(no inner side of canal of schlemm &
no spaces of Fontana) → long use of steroids
بسبب fibrosis

2-late cases : with corneal diameter ≥ 13 mm .

(canal of Sch → absent or fibrosed)

angle أقفل Do → External fistulizing operation e.g. trabeculectomy → استئصالها عامة



Either →(i) Alone, or (ii) With adjuvant use of Mitomycin C.
↓
inhibit mitosis

(tube أفرع) Setones ← لو فشلت كل المحاولات

Primary glaucoma

A) Closed Angle glaucoma

Definition : It is acute ↑of IOP due to sudden closure of angle

Etiology

(A) Predisposing Factors

(1) local : Narrow angle (commonly seen with shallow A.C) as in

(i) Hypermetropes (small eyes)

(ii) Old age (due to progressive ↑in lens thickness) → specially females (menopausal) → with hormonal replacement (Estrogen) → Congestion of vessels choroid تتأثر → affect middle coat & pushing iris forwards.

(2) General : Nervous individuals (with Imbalance between symp. and parasym, Tone) → Unstable vasomotor reaction → CB congestion → pushing iris forward.

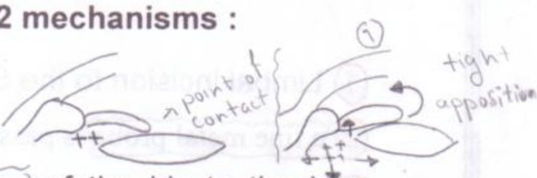
(B) Precipitating Factors :

- Pupillary dilatation due to → Mydriatics ,
  →Excitement,(بأتفرج على فيلم رعب)
  →or Prolonged stay in dark (قاعد في سيما)

Leads to angle closure through 2 mechanisms :

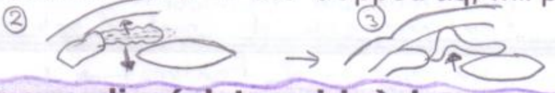
(lens & iris أهم المشكلات دائماً مشكلتين)

mechanism [illegible]



(i) Iris bombe (the most common mechanism)

In eyes with narrow angle , there is tight apposition of the iris to the lens → pupillary block that traps aqueous in posterior chamber . Dilatation ② of pupil → ② Relaxation of iris → the trapped aq, will ③ push the iris root forward → ③ angle closure .



mechanism [illegible]

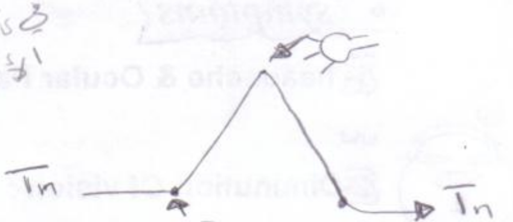
(ii) Iris crowding(plateau iris): less common

In which the root of dilated iris is crowded in angle occluding it → ↑ thickness of iris

N.B : The best proof of the 1 st mechanism is the curative effect of peripheral iridectomy .

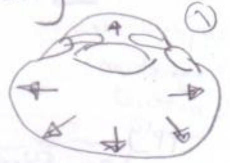
CL. Picture (stages) Oral & Written

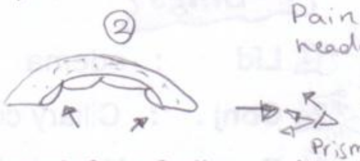
[illegible]



(I) Prodromal stage : (مرحلة الإنذار)

- This stage is characterized Transient attacks of increased IOP → P.D
- Transient attacks of : ① Headache , ∠45mmHg
  ② Blurred vision &
  ③ colored haloes around light
  (relieved by sleep or exposure to bright light)





بسبب Oedema

Diagnosis

In this stage , normal tension (in between attack) doesn't mean that pl. free & diagnosis depends on:

(i) History: → not migrain(① Blurred vision then ② headache)

(ii) Gonioscopy: Revealed narrow angle → (angle ضيقة لازم تتقفل) → لو لقيت wide angle يبقى احتمالك إن كنزها Prodroma إحمرار

(iii) Provocative tests

- ***Provocative tests***

* Idea: depends on pupillary dilatation .

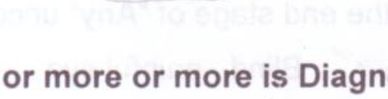
(a) Mydriatic lest :

- IOP is measured
- weak mydriatic is instilled e.g. phenyl ephrine 2.5% - 5%(not Atropine) → pupil واسع فا مش عاوزين أدخل المريضة في acute stage
- when the pupil dilates, IOP remeasured

* **Increase in I.O:P. by 8 mmHg or more is Diagnostic**

(b) Dark room test

- IOP is measured
- pt stay in a dark room for 1 hour
- IOP is remeasured



* **Increased I.O.P. by 8 mmHg or more or more is Diagnostic**

(لازم كل شوية الحكيمة تدخل عليها وتهزها علشان متنامش جوة الأوضة ويفشل الاختبار

(C) Dark room prone test:

As dark room but pt is in prone position.

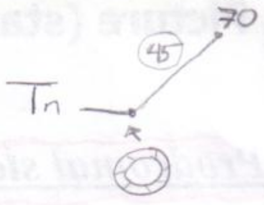
أحسن من dark room لأن prone position هيساعد على congestion فيهيئ
كل الظروف لحصول (Prodroma

Oral: Why pupil is vertically oval? Due to segmental ↑↑ tone in vertical fibers of dilator muscle.

(2) Acute (congestive) stage

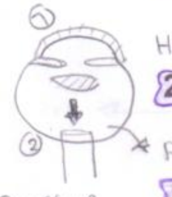
(المرحلة الأسوأ) Written & Oral very very imp.

• symptoms



1- headache & Ocular Pain: - severe &Bursting .

- & is referred to temple



HM

2-Diminution Of vision : due to -corneal edema

- Optic nerve ischemia

* PL (Perception of light

3- Nausea &Vomiting : due to reflex vagal stimulation.

① لو تعديت 45mmHg وحصل light stimulation برضه لا يمكن يقل

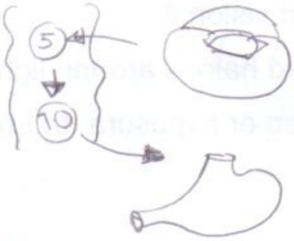
② طب ليه لا يزيد عن 70mmHg

← لأن الـ Pressure الموجود في Ciliary vessels ضغطه 150/70

علشان كده لو زاد IOP عن 70 V.C of vessels → ↓ Aq. formation → ↓ IOP

Pressure → compress veins → stagnation → ↓ arterial supply

لا ممكن يحصل الـقيء ↑↑ flood بسبب كل الـ 5th nerve



• Sings

1- Lid : edema

2- Conj . : Ciliary congestion

3- Cornea : Hazy (edema)

4- Ant.Ch. : Shallow

5- Iris : iris bombe

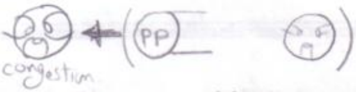
① no work of sph. pupillae.
② // // // dilator // .

6- Pupil : - Semi dilated , irreactive &Vertically oval

Paralysis of sph. pupillae

7- Fundus : hardly seen (due to corneal edema)

(A drop of glycerin on the cornea revealed Hyperemic & edematous op . disc) (Dark red)

(Acute congestive glucomal وده سبب تسميتو ←  PP)

• Fate :

The attack may - end in Resolution → with ttt

- Improve → to subacute or chronic stage

لو العيان كمّل ٢٤ ساعة وبعدين أخد (ttt ع) لازم يدفع تمن تأخيره

- Remain → leading to Absolute glaucoma

لو العيان كمّل وأخد analgesics وقال لما أفضى أبقى أروح للدكتور

(3) Chronic stage :

In this stage : long contact between iris & cornea → PAS formation .

Peripheral anterior synechia

ايه أخبار الـ triad بتاع الـ glucoma؟

- IOP : ↓but not to normal (due to PAS)(70mmHg → 30mmHg)
- Op. nerve: Show glaucomatous cupping
- Field : shows glaucomatous field defects .

(4) Absolute glaucoma

It is the end stage of "**Any**" uncontrolled glaucoma .

Symptoms : Blind , painful eye .

ملغي

~~***Signs*** : - Cornea : shows → Bullae (bullous keratopathy)~~

→ ~~its rupture → ulcer (↑pain)~~

→ ~~Degenerative pannus~~

- ~~Ant.Ch. : shallow~~
- ~~Iris : Atrophic patches - Ectropion uvea~~.
- ~~Lens : Complicated cataract~~.
- ~~Sclera : staphyloma~~
- ~~Fundus : Op. atrophy~~
- ~~Tension : T++~~

(5) Atrophic stage : Due to ciliary body atrophy . → by atrophy of epithelium (↓ CA enzyme & B receptors) → ↓ Aq. formation → ↓ IOP (70mmHg → 50mmHg → 20mmHg → 5mmHg

Treatment

Oral & Written
very very very Imp.

1- Prodromal stage :

As there are no PAS → peripheral, iridectomy is curative

(by allowing a way for aqueous from PC → AC)

لازم لازم تتحجز (وده أول خطوة لأن الحالة

2- Acute stage : دي emergency) ولازم دكتور النبطشي يسمع منك الكلام ده.

- Hospitalization .

ttt is essentially surgical , However, medical ttt is given for 24 hours to ↓ IOP

At the end of this trial , Gonioscopy is done

(i) If No PAS → do Peripheral iridectomy (in future → أكيد مش هيعمل iris bombe)

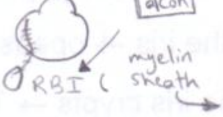
(ii) If PAS → do ext. fistulizing operation (Trabeculectomy)

3- Chronic stage : PAS are present, so do ext. fistulizing Operation .

4- Absolute glaucoma : This is blind , painful eye, so do

(i) enucleation . if refuse → (ii)

(ii) Retrobulbar alcohol



هيضرب myelin sheath of nerve → no sensation → no pain

(iii) Defunctioning op : C.B ضرب

cyclocryotherapy, cyclophotocoagulation, cyclodiathermy.

by freezing by lazer by heat

أشكال كل بتاعة RB.alcohol :

① Paralytic squint (by × 3, 4, 6th nerves)

② Proptosis due to retrobulbar hematoma

③ After 6 months → remylination of nerves → recurrent of nerve, وبعد كده الـ myelin هيعود على alcohol ويحصل الـ myelination بشكل أسرع كتير

N.B.

It is now clear that all patients with closed angle gl. must be operated upon. However, it is better (if we can) to do the operation after IOP is normalized. Operating on an eye with high tension carries risk of intraocular Hge (up to expulsive Hge), Lens sublux, or even dislocation.

Retrobulbar injection of alcohol :

Aim : to relief pain by destroying the ciliary ganglion. (myelin sheath بيذاب)

1 cc of novocaine 4% is injected followed by injection of 1 cc of alcohol 70%.

Complications: - (1) Retrobulbar Hge.

(2) Nerve lesion : with ptosis - squint – neuroparalytic Keratitis.

Oral:

أبتدي pilocarpine ولا mannitol ؟

(I) Medical Treatment

→ Start with Mannitol

↑ pressure > 45 mmHg → ↓ axoplasmic flow → paralysis of sp. pupillae → no change in pupil size → no effect of Pilocarpine.

(1) Local ttt

local ttt

a- Miotics (parasympathomimetics) → to act sphincter pupillae

Examples : pilocarpine nitrate 2-4%

1% weak

(lens مع iris [illegible])

>4% relative pupillary block due to contraction of circular fibers → ↑ curvature of lens after zonular relaxation.

Administration :

Every 5 min . until the pupil constricts ,then every 2-3 hours → till time of operation

Action :

1- contraction of sphincter pupillae ms leads to

- Pulls on the iris → opens the angle . ↑ Drainage

- widens the iris crypts → ↑ the drainage. ↑ Drainage

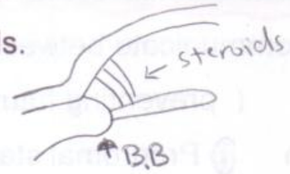
2- Contraction of ciliary ms leads to

- Pulls on the scleral spur → widens spaces of Fontana ↑ Drainage
- Compress the ciliary arteries → ↓ aqueous formation . ↓ formation

b- Other Topical Drugs:

1-Aqueous suppressant : as Beta blockers.

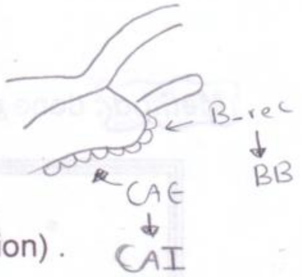
2-Anti-inflammatory : as topical steroids.



(2) General :

a- Carbonic Anhydrase inhibitors (CAI)

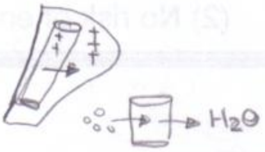
- Example : Acetazolamide (diamox Tab , I.V) (cidamex)
- Administration : 1 Tab (250 mg) /6 hours (I.V if vomiting) only 4 tablets
- Action : Inhibits aq. formation (by inhibiting the active secretion) .



b- Dehydrating agents (Hyperosmotic agents)

أحسن General +++

- **Examples :**



a- I.V. : Mannitol (20-25%)

dose: 1 gm / kg body weight, 250 ml تقريبا)

25 gm 100 ml

نفرض وزن الشخص ٧٥ كجم 75 gm 300 ml

b- Oral (1) glycerin (50%)

dose : 1 gm / kg Body weight

Drawbacks : i) sweet → nuseating (so it is given with lemon Juice)

ii) metabolized to glucose (→increased bl. Sugar) → مشكلة لمرضى السكر D.M

Action : Increases blood osmolarity

(i) ↓aq. formation (passive)

(ii) Draw H_2o out of eye (from the vitreous) .

Administration : Once for fear of :

- Brain cell dehydration (confusion)
- Circulatory overload (H.F)
- Urine retention in old pt .(in males due to prostatic hypertrophy)

Oral

الحل أقل ما أعطي العلاج ده لراجل مسن أركبله Catheter

c) Analgesics

→ Due to severe pain

d) Antiemetics

→ for nausea & vomiting

Surgical treatment

1- peripheral iridectomy

principle : to communicate between post. & ant. Chamber

(preventing future angle closure) .

Indications: in
(i) Prodromal stage .
(ii) Acute stage with no PAS
(iii) Prophylactic ttt in other eye .

Method : done either by (i) Surgery (ii) laser (Argon or YAG) (Thermal - effect)

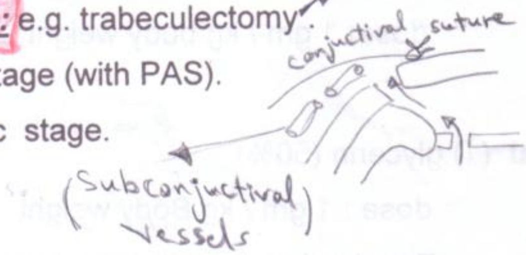
- ***N.B*** **:: Advantages of laser over surgery**

(1) carried out as an out - patient procedure (simple) (No hospitalization)

(2) No risk of endophthalmitis is or wound leak (↓ Hge)

(2) Externals fistulizing operation :

e.g. trabeculectomy + iridectomy بالمرة



Indications: in
(i) Acute stage (with PAS).
(ii) Chronic stage.

(3) Defunctioning operation :

Indications : in
(i) Absolute glaucoma
(ii) failure of fist. operations.

principle : decrease aq. formation by destroying the ciliary epithelium using :

- Freezing (cyclocryotherapy)
- Laser (cyclophotocoagulation)
- Heat (cyclodiathermy).

(4)Enucleation : in absolute glaucoma.

2nd cause of blindness in world

Open angle glaucoma

Definition :

It is bilateral, non congestive ↑ of IOP ③ in the ① absence of angle closure leading to optic n. ② damage & visual field defect .

Incidence :

- Age: above 50 ys
- Sex : equal .

N.B.

OAG is commonest type of Glaucoma:** ***- affecting 90% of gl . cases

- 2% of population > 50 ys .

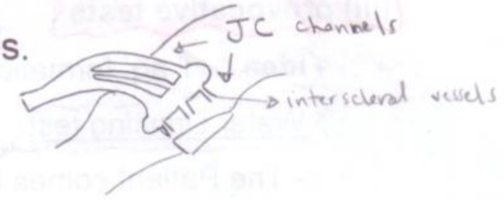
70% من الأنواع كلها كلها

Etiology :

Narrowing of aq. exit channels → ↓ aq. outflow due to :

-Sclerosis of T.M → narrowing of sp. of Fontana

-Sclerosis of the juxta-canalicular tissues.



Risk Factors:

1- Family history of glaucoma.

2- Age: more common above 50 years.

3- Dark races.

4- Ocular diseases: CRVO & Myopia.

5- Systemic diseases: DM & Vasospastic disorders: as migrane.

6- Ocular Hypertension.

Clinical picture :

المريض حظه أحسن لو
IOP → >30 mmHg
لأنه هيشعر ببعض الأعراض

Symptoms :

الأغلب ← (a) **Asymptomatic** : in some patients.

(b) **symptoms as :**

(1) Headache (mild but chronic)

Senile cortical cataract زي (2)Night blindness Due to peripheral field defects

(3) field defect or complete loss of vision in one eye (Pressure on optic nerve)

(II) Signs : (Written I.P.هم)

The 3 cardinal signs: High IOP - glaucomatous cupping - field defects .

• IOP :

↑ IOP above 22 mmHg in the presence of open angle is suspicious but not Diagnostic until it exceeds 26 mmHg ,however, Tn doesn't exclude OAG (LTG) . so to be sure we have to resort to the following methods :

Oral I.P.هم

(i) Tension in both eyes : normally, the difference doesn't exceed 2-3 mm Hg
A bigger difference (8 mm Hg) is more suggestive .

(ii)Diurnal variation : Normally, it doesn't exceed 2-3 mm Hg
if D.V. ≥ 8 mm Hg → OAG is suggestive .

(iii) provocative tests :

Normally tension isn't affected till the formation becomes 4 times.

- **Idea** : ↑ aq. formation (as drainage is not normal → the IOP will ↑)

* Water Drinking test

- The Patient comes fasting & IOP is measured, then
- The pt. Drinks one liter of H_2O (over 5-15 min) & tensions is remeasured.
- ↑IOP by 8 mmHg or more is Diagnostic

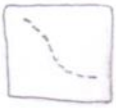
لا يشرب الماء دفعة واحدة بل على كدا كوب كل فترة (ربع ساعة تقريبًا)
→ to prevent reflex diuresis.

*Subconjuctival injection of priscol → VD of anterior ciliary artery → ↑ formation of Aq.

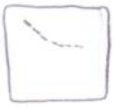
(iv)Tonography :

- An electronic Schiotz tonometer is continuously placed over the cornea for 4 min,
- The IOP is measured every 1/4 min (15 sec) and a curve is drawn.

from the curve the facility of aq. outflow is calculated .

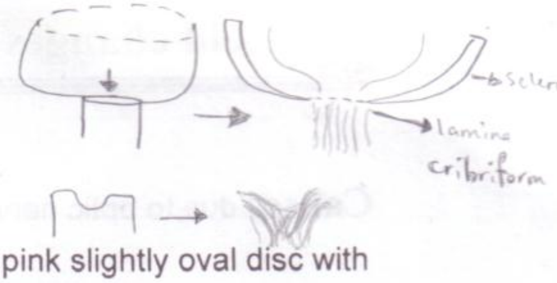


normal
spaces of Fontana
(Sloping Curve)

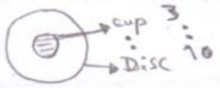


Problem in the angle (OAG)

• Glaucomatous Cupping



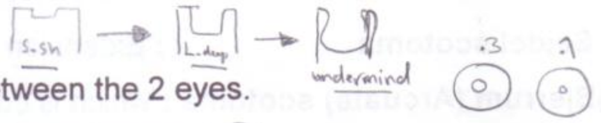
- The optic nerve head normally appears as a pale pink slightly oval disc with A central circular whitish depression with slopping edges called the physiologic cup
- Normal horizontal cup - disc ratio (C/D) = 0.3



- The optic cup in OAG is characterized by being :

Early:

1- large C/D (0.4).

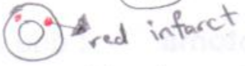


2- Asymmetry of C/D between the 2 eyes.

3- Vertical elongation of the optic cup.

بيكبر أولاً vertical وبعدين يـexpand في كل النواحي

4- Splinter Hge on the disk.



5- Increased visibility of the pores of lamina cribrosa.

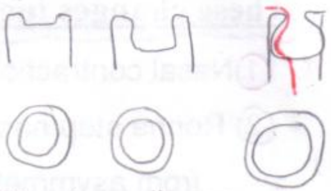
Late

1- large C/D more than 0.7. .

2- deep &

3- has over-hanging (undermined) edge with

4-the vessels appear as if interrupted.



- **Cause :**

(1) Mechanical theory : ↑IOP →backward bowing of lamina cribrosa .
(weak area in the outer coat) with mechanical pressure on the nerve fibers.

(2) Vascular theory : Sclerosis of disc vessels → n. fiber atrophy.

N.B. To detect disease progression the appearance of ONH should be recorded for future comparison by:

1- Optic disc photography.
2- Stereoscopic Photography.
3- Scanning laser Ophthalmoscopy.

Who dies first from retinal fibers?
① Most crowded fibers
→ T → N → Macula
② Most central photoreceptors
→ Part near to the edge.

• Field changes

Cause : due to optic nerve fibers damage

(I) Changes in the Central Field

- Central field : it is central 30° of the visual field .

The Changes include :

Scotoma: island of blindness

(1) Paracentral Scotoma : in the Bjerrum area (10-20 of fixation) .

(2) Seidel scotoma : Extension of blind spot above or below .

(3)Bjerrum (Arcuate) scotoma : which is continuous with blind spot & concentric with point of fixation . (Dying of arcuate fibers)

(4)Ring (Annular) scotoma : due to fusion of the 2 arcuate scotoma .

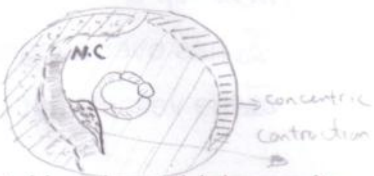
(II) Changes in the peripheral field :

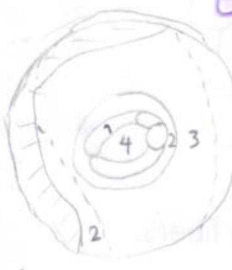
-The peripheral field limits : up & nasal → 60 °

- down → 70° - Temporal → 90°

-Testing (Perimetry) is done with : Goldman perimeter or Automated perimeter

These changes include :





①Nasal contraction(due to dying of temporal retina)

② Ronne step nasal defect with sharp horizontal border, which results from asymmetrical shape of the upper & lower field defects as they meet at the horizontal meridian

③Concentric contraction (more nasally)

④ Finally, central & peripheral changes meet → tubular field (+/- temporal island)

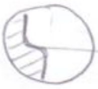
(If the macular area is involved → complete loss of vision) .

if 3&4 still working:

Wedge shaped appearance

N.B. For this reason vision is not a measure for the severity of OAG.

Oral: Ronne step nasal defect? ارسم



ماذا يمثل هذا الخط؟ على الـ Retina → raphe

ماشكله؟ خط مستقيم sharp ← ليه واحد سبق التاني؟

لعدم تساوي الـ receptors فوق وتحت الـ raphe.

إيه مشكلة OAG؟ إن آخر منطقة تتأثر في ال field هي ال macula وده
فالمريض لايشعر بأي مشكلة في ال field لحد ما نظره يضعف جداً ويبدأ يتأثر ال macula
تتأثر فيكون نظره في يوم 6/6 وفجأة في اليوم التالي (No perception of light)

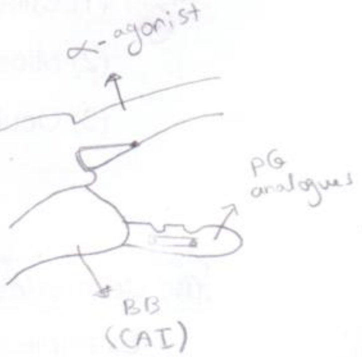
• Treatment

The ttt OAG is essentially "Medical" for life

(A) Medical ttt :

الترتيب (Written مهم)

1. B.B
2. B.B + topical CAI or PG analogue or α agonist
3. Miotics → في حالة اليأس في الآخر



(I) Local ttt:

(i) B- Blockers :

- Examples : Timolol maleate 1/4 - 1/2 %

  Betaxolol HCL 1/2%.

  Levobunolol ½%.
- Administration : every 12 hours .
- Action : decrease aqueous secretion (by 40%). ↓ formation
- Side effects :

  - Local : dry eye : punctate Keratitis
  - General : Bronchial asthma, Hypotension ,insomnia

    Depression, Impotence.

(ii) Prostaglandin analogues : → Some doctors prefer it than B.blockers because they have local side effects only not as B-blockers

- Examples : Latanoprost (Xalatan).
- Administration: once daily.
- Action: increase the uveo-scleral outflow.
- Side effects: يلا خليها سكانة أوروبا

  - Iris : iris pigmentation, iritis.
  - Lid: increased lid pigmentation, overgrowth of lashes.
  - CME in aphakics & pseudophakics.

    Cystoid nuclear edema

(ii) Alpha Agonists :

Side effects:

① Pigmentation of lower fornix (by epinephrine)
② Tachycardia ♡ ↑↑

- Example : adrenaline 0.5-1% (obsolete)

  Dipivefrine (propine)0.1 %

  Bromonidine(alphagan)
- Administration : every 12 hours
- Actions : (1) aq. outflow (widens spaces of fontana) (2) V.C of C.B vessels .

(iii) Miotics :

- examples : Pilocarpine 1-4 %
- Action : see before 4 – opening angle because the angle is already opened
- Administration : 3-4 times daily / for life → miotic life .
- Side effects :

relaxation of zonules → ↑ curvature of lens

Maintained contraction

(1) Ciliary spasm → headache & fluctuating myopia (1-2D) Myopia occurs only after drops.

(2) Miosis → night vision &marked diminution of vision in nuclear cataract.

due to Presence of day blindness

(بعد القطرة يا دكتور مش شايف أي حاجة قدامي)

(3) Ocular congestion → discomfort .

(iv) Carbonic Anhydrase inhibitors :

-Example: dorzolamide.

-Administration: every 12 hours

35 mmHg
↓
Med ttt
→ 15 mmHg → follow up
→ 24 mmHg → [illegible] Laser Trabeculoplasty
→ 30 mmHg → لازم Surgery

(II) General ttt

Carbonic Anhydrase inhibitors :

- Examples ,administration & action : see before
- side effects (1) Hypokalemia → parathesia of fingers & toes K↓

لايمكن يحصل في closed angle glucoma لأن المريض بيأخذه لمدة ٤٨ ساعة فقط

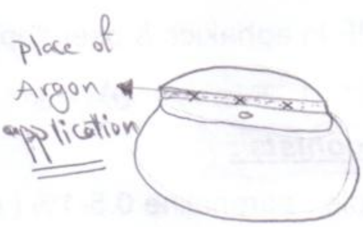
(2) leucopenia & aplastic anemia: due to bone marrow depression . ↓B.M

(3) Renal stones . ox ↑

Follow up at regular intervals (Tension - disc – field) is essential.

(B) Laser Trabeculoplasty :

Technique:



1- Topical anesthesia & miotics.

2- Application of gonioscope.

3- Application of one or two rows of low powered laser (argon or YAG) to TM

Principle : not clear : May be by inducing minimal scaring at T. meshwork → its contraction → widening of spaces of Fontana.

Indications : Mild to moderate cases (IOP < 35 mmHg) ,used with medical ttt.

(C) Surgical Treatment :

Indications : if

(1) Medical ttt fails to control IOP.

حتى لو كن IOP ↓ 15 mmHg

(2) Deterioration of the visual field (In spite of IOP control)

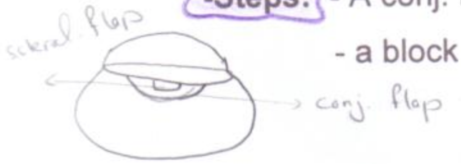
(3) The pt is poor , Negligent or cannot be followed up . In Egypt only

Principle → Filtering operation (subscleral Trabeculectomy) + Iridectomy

- Aim : producing a fistula between the A.C. and the subconjunctival space where aqueous is absorbed by subconjunctival blood vessels .

-Steps: - A conj. Flap is made. – A scleral flap is raised.

- a block of T.M is excised . - Closure of the scleral and conj flaps.



اقصر سمك
[illegible] sclera

ملغي

~~*N.B.* Internal fistulizing operation (cyclodialysis)~~

~~**Principle** : to communicate (fistula) between A.C & suprachoroidal space where aq. is absorbed by choroidal blood vessels~~

~~**Indication** : in aphakic glaucoma (as , vitreous may herniate & block the ext. fistula)~~

ملغي

~~Hypotony~~

~~**Definition :** ↓ IOP below 10 mmHg~~

~~**Etiology :**~~

~~(I) Local causes:~~

~~(i) ↓ Aq. Formation: -post - inflammatory (cyclitis)~~

~~- post - traumatic (C.B shock)~~

~~(ii) ↑ Aq. out flow : corneal fistula - wound gap - R.D .~~

~~(II) General causes :~~

~~(i) Diabetic coma (ii) Uremia (iii) dehydration~~

~~**Complications :** IOP → V.D → transudation → Retinal & disc edema~~

Secondary glaucoma

Definition : It is increased IOP secondary to a local eye disease

Etiology

(I)Corneal causes:

Open angle:

Corneal ulcer : due to : plasmoid aq.

Closed angle

(1) Leucoma adherent & Ant staphyloma : due to : PAS (CA).

(2) Corneal Fistula (after its closure): due to PAS (CA)

(II) Ant. Chamber causes

Open angle:

(1) plasmoid aq., Hypopyon & Hyphema (OA).

(2)angle recession glaucoma : due to tears in anterior surface of CB.

Closed angle

(1) Epithelial cyst .

(2) Ant. Lens dislocation (due to pupillary block).

(III) Iris causes :

Open angle:

(1)Acute iritis : due to plasmoid aq. , hypopyon (OA) .

(2) Glaucomato-cyclitic crises: unilateral rise of IOP

(cause: may be disturbance of prostaglandin metabolism).

(3) Herpes S.& Z. : due trabeculitis

(3) Rubeosis Irides : Early.

Closed angle

(1) Chronic iritis (CA) :due to seclusio or occlusio pupillae (CA).

(2) Rubeosis Irides : Late

P10 Oral: What is Phacogenic glucoma?

(IV) lens causes :

Open angle:

① **Phacolytic gl (Hypermature cataract)** : Where the leaking lens ptn were engulfed by macrophages which in turn trapped in TM.

② **Phaco-anaphylactic Glaucoma** : It follows trauma or surgery , where lens ptn are released into the ocular fluids and initiate an auto-immune reaction , the products of which are deposited in the trabecular pores.

③ **Subluxation or post dislocation** : due to iritis (OA) anterior or posterior dis by herniation of vitreous.

④ **Pseudo-exfoliation glaucoma** : due to excessive production of basement membrane like material by the ocular epithelium, resulting in deposition of dandruff-like material in the TM leading to its obstruction (not as exfoliation)
↓
difference source of dandruff

Closed angle

① Intumescent cataract (Phacomorphic gl.) :due to pupillary block

② Ant. dislocation due to pup block (gl inversus)

③ Post. Dislocation or Subluxation :due to pup. block by the vitreous

ttt : Removal of the lens

Aphakic Gl. :

It is increased IOP : Following cataract extraction Due to :

(1) post - operative iritis (manipulation retained lens matter)

(2) post - operative hyphema .

(3) pupillary block by vitreous, air, IOL.

(4) Steroid induced Gl.

(IV) Retinal causes :

(1) Diabetic retinopathy & CRVO: due to : retinal ischemia → Rubeosis iridis

(2) Ret. detachment surgery : Due to :

(1) tight scleral buckle

(2) Intraocular injection of gases or silicone oil

(3) Steroid ttt

(VI) Intraocular tumors

Mechanism : 1) Space occupying lesion.
2) Pushing the iris - lens diaphragm forwards .
3) Intraocular Hge.
4)Iridocyclytis : Hypopyon , Hyphema.
5) Closure of the angle by malignant cells.

(VII) Drugs :

(1) Steroids (OA) (2) Mydriatics (CA)

(VIII) Trauma

due to (1) hyphema (2) Corneal ulcer & iritis
(3) Angle recession gl. (4) Siderosis bulbi : IOFB (iron)

(IX) Miscellaneous :

(1) Malignant Glaucoma

- **It follow** intraocular surgery particularly cataract & glaucoma surgery.
- **Mechanism :** a cilio - vitreo lenticular block resulting in misdirection of aq. posteriorly. This misdirected aq. pushes the vitreous forwards aggravating the block.

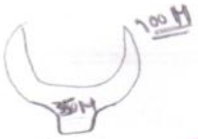
Retina

Anatomy

Gross

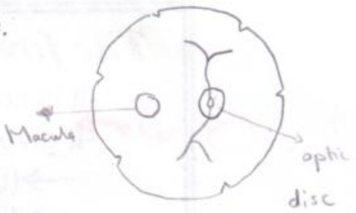
- مكانها ➢ It is the innermost layer of the eyeball.
- حدودها ➢ It starts at ora serrata & ends at optic disc.
- خصائصها ➢ It is very thin & transparent showing the red color of choroid.
- وظيفتها ➢ It is sensitive only to light (translates photons to electrical imulses that reach the visual cortex and transformed into a visual sensation).
- ➢ **Anatomically**, the retina is divided into :

  1- Central Retina :(Macula lutea = 5 mm) The center of the macula is an avascular depression in its center called " fovea centralis (1.5mm)

  Function: (Mainly cones)

  Responsible for Visual acuity, Color vision, Form sense.

  2- Peripheral Retina: ends at the ora serrata

  Function : (Mainly rods)

  responsible for night vision and peripheral field.

Microscopic

The retina is composed of 10 layers

(1) Pigment epithelium:

- ➢ Its is outermost layer (in contact with Bruch membrane of choroid).
- ➢ It is made of one layer of pigmented cubical cells.

(2) Photo – receptors (rods & cones):

- ➢ The light sensitive layer.

(3) Outer limiting membrane:

- ➢ It is made of the ends of Muller's fibers.

(4) Outer Nuclear layer:

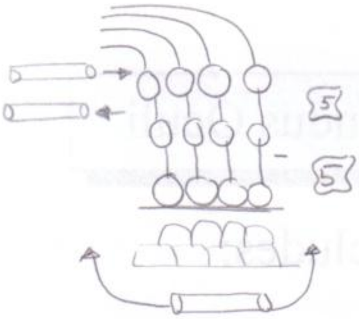
- ➢ It's made of nuclei of rods & cones.

(5) Outer plexiform layer :

- ➢ Made up of the synapses between the outer &inner nuclear layers.

(6) Inner Nuclear layer:

- ➢ It is made up of bipolar cells & cells of Muller's fibers.



Blood Supply:

(1) Arteries:

- Inner 5 layers: from central retinal artery (from the ophthalmic a.)The CRA pierces the subarachnoid accompanied by the the vein to reach the center of the nerve . It turns forward at the right angle and passes through the lamina cribrosa to enter the physiologic cup.

- Outer 5 layers: avascular (nutrition by diffusion from the choroid)

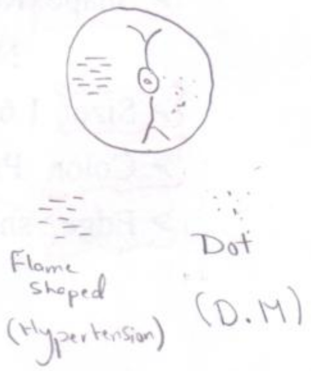
(In 20% of normal people: a separate artery originating from one of the posterior ciliary arteries supplies the macula called cilioretinal artery).

(2) Veins:

The retina is drained into the central retinal vein (CRV).
(to the superior ophthalmic vein or directly to the the cavernous sinus).

Retinal Capillaries

- Superficial plexus (in nerve fiber layer) : Hge in this plexus take distribution of nerve fiber layer → flame shaped
- Deep plexus (in the inner nuclear layer) : Hge from this plexus → rounded (dot) hge.



The Fundus Oculi

- Includes:

1- The Retina 2- The optic Disc (the visible part of optic nerve)

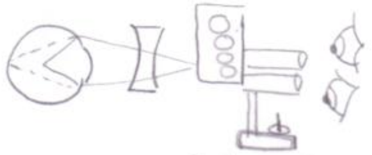
3- The choroid 4- The vitrous.

- ***Examination of the fundus: by*** Oral: why you use?
  Both → 1st: Screening by 2 → flat or elevated lesion → [illegible] lesion
  2nd: By 1 to see lesion better

(1) Direct Ophthalmoscope (from retina of Pt to retina of examiner directly)	(2) Indirect Ophthalmoscope (2nd focus on retina of examiner)
Erect Image	Inverted image
Magnification :X 15	Magnification : X5
Field smaller	Field larger
Uniocular	Binocular
No Stereopsis	Stereopsis(Binocular vision) → صورة 3D مجسمة

الأفضل **(3) Slit lamp bimicroscopy: with a fundus lens (+90 or +60 D.)**

- ➢ Advantages: high magnification (by lens), and stereopsis (by binocular vision)
- ➢ Disadvantages: Inverted image.

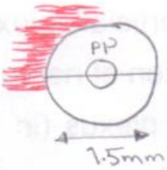


يتحرك للأمام و للخلف

Appearance of Normal Fundus

Oral

(1) Optic Disc:



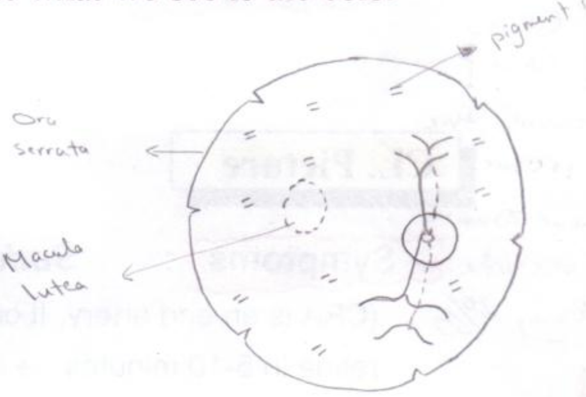
- ➢ Site :Nasal to the posterior pole of the eye
- ➢ Shape: Rounded with physiological cup in its center.
  Normally C/D = 0.3 (from which the central vessels emerge)
- ➢ Size : 1.6 mm (in diameter)
- ➢ Color : Pale pink
- ➢ Edge : sharply defined (so, used as unit of measurement)
  By its colour

(2) Macula lutea :

- **Diameter** : about 5 mm .
- **Site**: its center is 2 D.D temporal & slightly below the center of disc → شوية تحت إزاي من شوية قلك بتحدد [optic disc → as unit of measurement.
- **Color**: Yellow-red due to high content of xanthophil pigment.
- **Fovea centralis**: - is a small dark red depression in its center (1 DD)
  - showing a bright foveal reflex.

(3) Retinal Vessels :

- The CRA emerges from .cup & then divide into upper & lower branches. These branches subdivided into nasal & Temporal branches
- The veins follow the arteries (Arterio-venous crossing)
- Arteries are smaller than veins (A/V = 2/3)
- The retinal vessels are transparent tubes & what we see is the color of blood through them.



(4) General background :

It shows : Tigroid fundus (خلفية حمرا منقطة بأسود)

- Choroidal vessels (red areas)
- Choroidal pigment (dark areas)

(5) Ora Serrata.

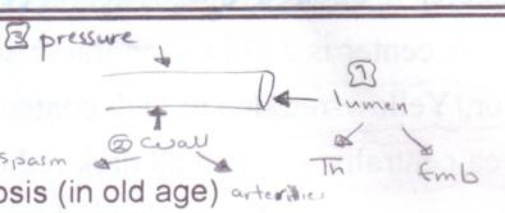
Diseases of Retina

(1) Vascular **(2) Ret. Detachment** **(3) Degeneration**

Vascular Diseases

- Vas. Occlusion (CRAO & CRVO)
- Vas. Sclerosis & retinopathies

Central Retinal Artery Occlusion



Etiology

Oral

- **Thrombosis**: In atherosclerosis (in old age) arteritis
- **Embolism:**

  a- Detached thrombus: as in

  -Myocardial infarction , aortic aneurysms or subac. B Endocarditis

  b- Air Embolism.

  c- fat Embolism

  as in - Crush injury. – Acute hemorrhagic pancreatitis
- **Spasm**: as in - Hypertensive Encephalopathy → in young age → infarction

  - Quinine poisoning - Migrane

  لأن لو حصل
  انسكاب، نزيف
  طوالي Hge
- **Arteritis**: as in - Giant cell arteritis - Poly arteritis nodosa
- **Very high IOP**. as during RD surgery → through large gas bubble that compress the artery

Oral
Can glucoma causes CRAO at any case?
No, because the highest pressure can occur 70 mmHg can't occlude the artery 80/60

CL. Picture

- **Symptoms** : ***Sudden Painless loss of vision***

(CRA is an end artery; It occlusion → coagulative necrosis of the inner ½ of retina in 5-10 minutes → complete loss of vision)

-Sometimes, if the macula is supplied from the cilio -retinal artery (in 20% of population) → Central (tubular) vision is preserved.

Oral
Can Pt see 6/6 with CRAO?
Yes, in 20% of cases but with tubular field

End artery → No anastmosis

- **Signs**

(1) Vision → No P.L

(2) Pupil : → No P.L within seconds.

- **Direct reaction** → absent - **Consensual** → preserved (= afferent pupillary defect)

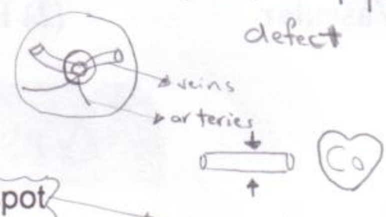
(3) Fundus

Oral
very very important

(i) Early

- Arteries : attenuated (thread – like).
- Veins : little attenuation .
- Retina : Center →shows cherry red spot

Why?
No musculosa to squeeze blood So, it becomes stagnant.



باين جداً لأن
حوله لون أبيض
مش زي الأحوال
الطبيعية

(ii) late

- Arteries : refill : due to - relief of spasm . -
  - canalization of the thrombus .
- Retina : the cherry red spot disappears
  (as the macrophages engulf the dead ganglion cells).
- Disc : shows Consecutive optic atrophy : → due to cutting of blood supply by the brain.

N.B (Oral very very important) Cherry RS **: appears 2-3 hours form the onset of occlusion**

(i) Milky white :due to Coagulative necrosis in ganglion cell layer

(ii) Fovea Contains no gang. Cells → remains red (showing choroid).

D.D

** Other Causes of cherry red spot

- *Commotio Retinae*
- *Quinine poisoning*
- CRAO
- *Amaurotic family Idiocy*
- *Macular hole surrounded by R.D .*

Treatment

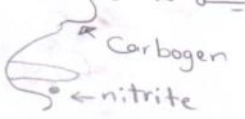
الحل الوحيد للمشكلة

إن اللي يجيله CRAO يبقى دكتور رمد فيعرف على طول
اللي جاله وأخوه دكتور رمد علشان يعالجه ويبعتوا
الاتنين ساكنين فوق المستشفى.

- Usually Useless (if not immediate) → within 30 minutes.
  - Aim is to induce Vasodilatation

    If spasm : :relief the spasm.

    If embolus : dislodge the embolus to a smaller branch)



By • **General** : as 1- inhalation or sublingual of amyl nitrite .

2- Inhalation of carbogen. → (sudden & severe V.D) → Due

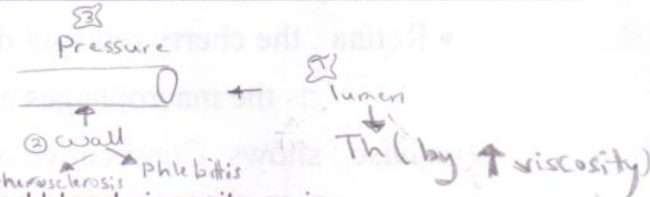
3- I.V. acetazolamide (500mg) → ↓ IOP → VD of vessel.

4- I.V. streptokinase

• **Local :** - Paracentesis (leading to Hypotony -----V.D)

- Ocular massage to lower the IOP.

Central Retinal Vein Occlusion



Etiology

1- *Inside the vein*: due to increased blood viscosity as in :

Polycythemia, Leukemia – Dehydration.

***2- In the vein wall* :**

- **Phlebitis** as in typhoid, influenza (young).
- **Rough intima** as in atherosclerosis (old) & DM.

3- *Outside the vein*: Due to pressure on vein by

orbital tumor- increased IOP - sclerosed artery.

very rare

most common (common site → (Arterio-venous crossing)

Clinical Picture

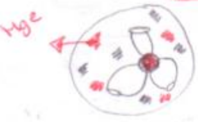
➢ Symptoms

زغللة بسيطة ← زيادة
في الزغللة ← H.M
(loss of vission فقد
مرة واحدة)

Rapid of ↓ vision

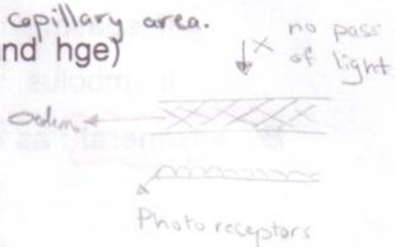
(usually observed at the morning as the thrombus forms at night)

➢ Signs :



→ weak response inspite of strong stimulus.

- **Pupil :** sluggish reaction (due to retinal edema and hge)
- **Fundus :** shows stormy sunset appearance
  - **Retina :** show Hge, exudate, edema
  - **Disc :** Hyperemic, edematous
  - **Veins :** engorged & tortuous



Oral:

Complications

1- Neovessels formation :

due to retinal ischemia→ release of vasogenic material

- **Site :** Retina (NVDs & NVE) – Iris (Rubeosis iridis)→ vessel ظهر iris على ويتحول لونه أحمر
- **These vessels may rupture** Hyphema – vitreous Hge

No foveal oedema as no vessels

2- Macular Edema (up to cystoid macular edema → macular scar)

This leads to ↓ vision

3- Secondary Glaucoma : due to

- **Organized blood in angle** (Hemorrhagic glaucoma).
- **Neovessels in angle** (Neovascular glaucoma):
  - Occur in 20% of patients → As they suffer from ischemic
  - Takes about 3 months to develop (100 day glaucoma)

أسوأ مشكلة → Due to failure of fistulizing operation (rupture vessels → when hyphema treated → formation of new vessels) (مفيش حل)

Oral

هام جداً

(الفترة ما بين occlusion و ظهور complications)

Classification

(1) Ischaemic **(2) Non-ischaemic** **(3) Papillophlebitis.**

> 80% < 20%

	Non-ischaemic	Ischemic
Site of obstruction	Post. To L. Cribrosa (good collaterals)	Anterior to L. Cribrosa (No collaterals)
Incidence	More common	Less common
Symptoms	Blurring of vision	Marked ↓ of vision
Hge, exudate	Mild Hard	Extensive Soft (retinal infarction)
Complications	Rare	Common
Prognosis	Good	Poor

D.D.

مهم

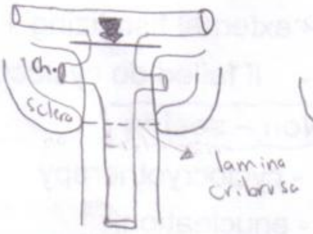
- **From other causes of retinal Hge**

a) General cause

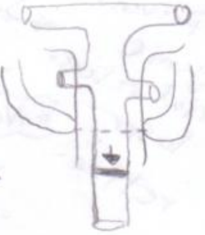
(1) Blood diseases (e.g. leukemia)

(2) Vascular diseases (as hypertension)

(3) sever straining



Ischemic



Non-ischemic

b) Local causes

(1) Trauma (2) Hypotony. (3) Tumors (4) Diab. Retinopathy

سؤال

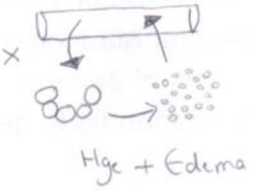
• From other causes of rapid ↓ of vision

(1) Acute glaucoma (tension)

(2) Commotio retinae (history of trauma)

(3) Uraemic amaurosis (renal failure).

Treatment



(1) Medical

- ttt of the cause
- Help blood absorption : alpha – chymotrypsin.
- Prevent Platelets stickiness : Aspirin in small doses (75 mg).
- Anticoagulant : may cause Hge (not used)
- Vit C → to preserve integrity of wall of blood vessel (Prevent Hge)

(2) Laser Photocoagulation (argon) → not YAG (cutting)

Indication: in Ischemic cases (after the hge resolves)

Aim :

- To destroy the hypoxia areas (→ anoxic) no vasogenic material releases.
- Regression of the neovessels. (retraction of vessels which where present)
- Improvement of circulation of the central area. (أخذت الدم اللي كان رايح periphery)

Normally

Centre: 60% of blood supply.

Periphry: 40% of blood supply

(3) Ttt Of Neovascular glaucoma:

• Seeing eye

- Medical : B.B Timolol + Diamox + Steroid (locally)
- Laser ttt
- external fistulizing + Implant (Ahmed valve or Molteno tube)
- If failed do cyclocryotherapy.

& prevent vasogenic material release by laser to retina.

× BB + CAI + steroid + Atropine

• Non – seeing :

- cyclocryotherapy
- enucleation.

إياك تحطيه Miotics → Constric vessels → Congestion in new vessels which are fragile (مصيبة)

Vascular Sclerosis

The arteriosclerosis changes in the retinal vessels are

(1) Changes in light reflex : - Copper wire → ↓luministy of red colour due to fibrosis . - Silver wire → More fibrosis.

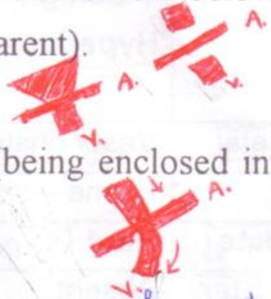
(2) Sheathing of vessels (by blood in centre & greyish fibrosis in periphry)

(3) Change in : - Course : tortousity **- Diameter :** irregular → by fibrosis

(4) Arterio – Venous (A/V) crossing changes

Normal

Oral Tip.



- ***Gunn's sign (Nicking)***: Obscuration of a part of the vein below the arterial wall (that becomes not transparent).
- ***Banking*** : Distension, of vein distal to crossing. (الدم متجمع في منطقة واحدة)
- ***Deflection*** : of course of vein to disc by rigid artery (being enclosed in one sheath)= (Salus sign)

Vascular light reflex Reflection of light from apex of vessels due to maximum convexity

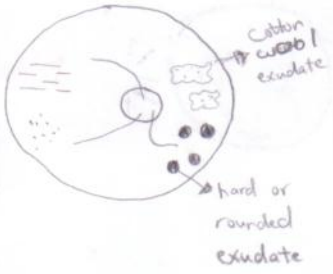
Vascular Retinopathy

Definition

It is bilateral retinal affection 2ry to systemic vascular Disease. (As hypertension)

(أهم كلمة في الأسئلة ← systemic)

Cl. Picture



Fundus examination shows 3 Ophthalmoscopic signs

1. ***• Changes in the Blood Vessel:***
   - Vasoconstriction (sclerosis or spasm).
2. ***• Retinal Hge :***
   - **Superficial** : Flame shaped (in the nerve fiber layer).
   - **Deep** : Dots (in the inner nuclear layer).
3. ***• Retinal exudate :***
   - **Soft (Cotton wool) exudate** → not exudate :

     White ,large , ill-defined patches (retinal infraction)
   - **Hard exudate** :

     Yellowish white , small ,rounded ,deep spots with well-defined edges (hyaline degeneration with lipid deposits)

- **Symptoms:**

① Mild cases : Asymptomatic

② Severe cases :- Metamorphopsia & blurring of vision (M. edema)

- Rapid ↓ of vision (due to exudative RD)

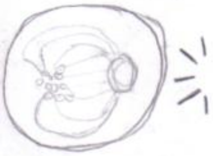
diminution

*** Signs : Fundus Picture**

Oral

	1-Benign Hypertension (old age)	2- Malignant & Renal Hypertension	3-toxemia of pregnancy
Vessels	Vasoconstriction due to (atherosclerosis)	Vasoconstriction due to (↑x3)	Spasm ---Ischemia
Hge	Flame – shaped	Flame shaped	Flame shaped
Exudate superficial	Hard (chronic ↑)	Soft (Sudden ↑)	Soft (Sudden ↑)
Mac. Star	Absent	Present	Present
Retinal Edema	Absent or mild (mild ↑↑ in pressure)	Present (in the retina & in the disc)	Present (retina & disc)& up to ex.R.D
Significance	Pt. is liable to cardiac & cerebral accidents Medical care is in need	Pt rarely lives more than 2ys. death is usually due to renal Failure.	Termination of pregnancy must be done to save life & vision of mother

Macular star: Exudate at macular area & this appearance is due to distribution of fibers around fovea (Henle's layer)



Macular fan:

Diabetic Retinopathy

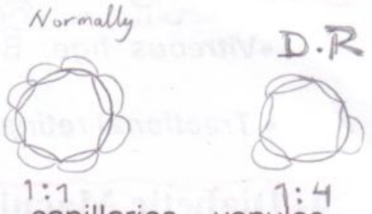
Definition

It is bilateral retinal affection due to DM

Etiology

Unknown

Pathogenesis

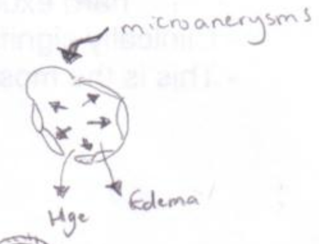


DR is a micro–angiopathy affecting the retinal arterioles – capillaries – venules

- ***Micro – vascular leakage :***

  1- Due to : loss of pericytes.

  2- Resulting in (Edema – Hge – micro aneurysm).

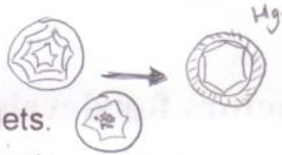


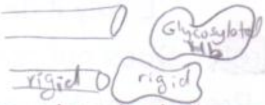
- ***Micro – vascular occlusion***

  1) Due to

organic occlusion
1- Thickening of basement memb.
2- Aggregation & stickiness of platelets.
3- Endothelial cell damage & proliferation.

functional occlusion
4- RBCs changes (Glucosylated Hemoglobin & lack of deformability). (can't slide itself)





2) Resulting in :

Retinal ischemia which release chemical (vasogenic) factor which stimulate new vessels formation.

Clinical picture

(Oral [illegible])

1- Non –Proliferative (Simple or Background) DR :

(Don't affect vision → 6/6 [illegible])

- ***Retinal Hge :*** Deep (rounded hge)
- ***Retinal exudate :*** Deep (hard) exudate & edema
- ***Retinal vessels :*** Micro – aneurysm (seen by FI. ~~Angiopathy)~~ Angiography)
- ***Macular edema*** : up to CME.

2- Pre-proliferative

→ Phase of newvessel formation only.

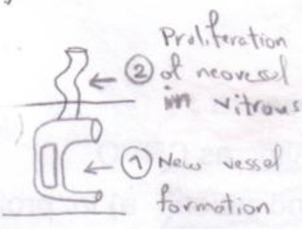
- ***Retinal Hge :*** Dots & blots.
- ***Retinal exudate :*** Deep (hard) exudate & edema
- ***Retinal vessels :*** IRMA & venous beading &looping (Intra-retinal microvascular abnormalities)
- ***Macular edema*** : up to CME.
- ***Retinal Ischemia:*** (detected by Fl. Angiography)

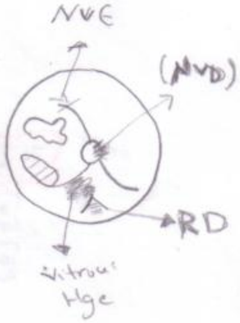
3- Proliferative DR :

الأسوأ

Proliferation of neovessel in vitrous

- ***Retinal hge :*** Blots (Due to rupture of large neovessels)
- ***Retinal exudate :*** Cotton wool
- ***Retinal vessels :*** neovessels (NV) at
  - 1- The Disk → NVD → neuvessel at disc
  - 2- The Retina → NVE (elsewhere)





Oral: Why marked ↓ of vision in Proliferative D.R?
Due to ① Vitrous Hge ② Tractional R.D

- ***Vitreous hge:*** Bleeding from the neovessels, which may organize → vitreous fibrosis
- ***Tractional retinal detachment:*** Due to pull by the vitreous fibrosis.

4- Diabetic Maculopathy: either

- Diabetic maculopathy is the presense of any retinal thickening or hard exudate within 1500m of the center of the fovea (Central)
- Clinically significant edema : if the edema or exudate is within 500m.
- This is the most common cause of diminution of vision in diabetic pts.

Oral مهم

N.B. Risk Factors for developing DR

1- The duration of DM : the longer the duration of DM , the more Likely it develop retinopathy.

2- Metabolic Control : Does not prevent, but delays the onset of DR.

3- Other factors: Pregnancy, Hypertension,Anemia, Renal disease & smoking have an adverse effects onDR.

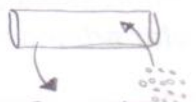
D.D.

1- ***From other causes of retinal hge :*** see before.

2-***From other causes of vascular retinopathy*** : see the table.

Treatment :

1- Medical ttt : (background) ×



يؤجل المشاكل ولا يلغيها

- Control DM & hypertension.
- ↓ Platelet stickiness → Aspirin (in small doses)
- Fasten absorption of → 1-exudate → give Doxium
  → Hge → Alpha-chymotrypsin & vitamin C

2- Laser ttt :

- Aim : as CRVO.
- Indication : a) In proliferative DR do PRP.(Pan-retinal-photocoagulation)
  &b) In diabetic macular edema do focal ttt. (F↓↑)
  laser

↑ for vitrous Hge

3- *Vitrectomy :* + endolaser

- Aim
  1- Clears vitreous hge, especially if :
  - Recurrent.
  - Associated with Rubiosis iridis (to prevent NVG)

  2- Removes vitreous fibrosis → prevent & treat tractional RD.
- Indications: proliferative DR.

4- ***Neovascular glaucoma*** : as in CRVO.

N.B. Regular fundus examination of diabetic pts is essential.

Oral (هام جدا جدا جدا)

**Ocular manifestations of DM

(1) **Lid:** blepharitis, recurrent styes.

(2) **Conjunctiva:** conjunctivitis.

(3) **Cornea**: Keratitis (ulcer).

(4) **AC:** Hyphema (From rubeosis iridis)

(5) **Iris:** Rubeosis Iridis.

(6) **Lens:** Complicated Cataract

(7) **Changes in refraction**: a) Hyperglycemia → Myopia.
b) Hypoglycemia → Hypermetropia.

(7) **Vitreous:** Vitreous hemorrhage (From neovessels in DR)

(8) **Retina:** - DR - CRVO (→ Retinal hge)
- Lipemia retinalis: due to hyperlipidemia (showing Pale fundus & milky vessels)

(9) **IOP** : **May** : - ↑ : as in Neovascular & haemorrhagic glaucoma.
- ↓ : Hypotony in diabetic coma.

(10) **Optic nerve** : Optic neuritis.

(11) **Orbit** : Orbital cellulitis.

(12) **Extraocular muscles:** Paralytic squint (specially the Lat. Rectus)

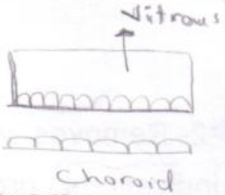
(13) **Post operative complications** : Hge, infection, delayed wound healing

Oral Marked drop of vision in D.M?
→ Due to all previous causes except causes → lid
→ Conjuctiva.

Separation of retina between RPE & photoreceptor layer is due presence of potential space between ① outer wall of optic cup giving RPE ② inner wall of optic cup giving sensory retina.

Retinal detachment

Definition

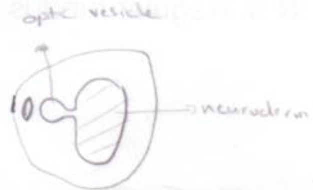


It's a condition in which the retina is separated into 2 layers :

(1)- Retinal pigmented epith. (R.P.E.)

(2)- Sensory retina (9 layers)

So, the term retinal separation is more accurate

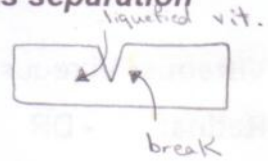


Etiology (Types)

break

(A) 1ry (Simple – Rhegmatogenous)

- ***It's due to formation of a retinal break, which allow the liquefied vitreous to enter between the layers of the retina & cause its separation***



• N.B.

1- Retinal breaks :

are full-thickness defect in sensory retina & are either

1- Retinal Holes : without any traction

Cause :chronic atrophy of sensory retina (less risk of RD).

Shape : round or oval.

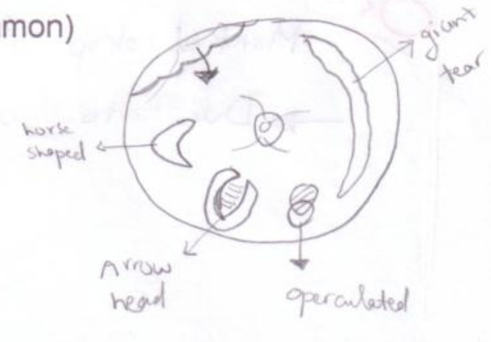
2- Retinal Tear : with traction.(but not tractional because it is not due to fibrosis)

Cause : vitreo-retinal traction..

Shape : (i) Horse shoe tear (the most common)

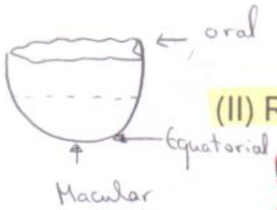
(ii)Arrow head tear .

(iii) Operculated tear.



(iv) Disinsertion (retinal dialysis): Separation of the retina From its root at the ora serrata.

(v) Giant tear : 90 degree or more of globe circumference.



Site : - Oral, -Post-oral, - Equatorial , Post-equatorial - Macular

(II) Risk Factor : of Rhegma. RD :

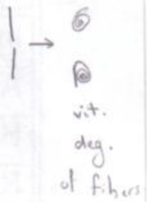
- (a) Chorio – retinal degeneration as in High myopia. (separation between collagenous & liquefied Parts of vitrous)
- (b) Blunt trauma.
- (c) Aphakia
- (d) Family history of RD or history of RD in the fellow eye.

Clinical Picture of rhegmatogenous RD

• Symptoms:

1- Early:

- **Flashes of light (photopsia) :** Due to irritation of rods & cones by vitreous traction at sites of vitro-retinal adhesion. (Because single response of receptors is light)
- **Floaters: (Musca volitans):** Due to Vitreous Degeneration or hge.
- **Macropsia**⑤**, Micropsia**②**, Metamorphopsia**③**(** Distortion of objects).



2- late:

- **Field defect (black curtain):** its site is useful in predicting the Site of the retinal break (in the opposite quadrant).
- ↓ **Of vision :**due to foveal detachment.

• Signs :

1- R.R : Grey. Hazy retina which obscure choroid

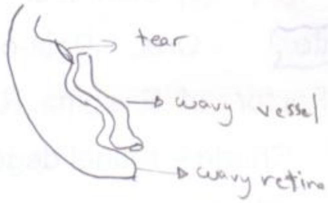
2- Tonometry : Soft tension

(Due to absorption of the SRF by the choroidal vessels).

3- Slit lamp : may show Flare (if Iridocyclytis occur).

4- Ophthalmoscope : Fundus examination shows :

- Retina → wavy.
- Retinal vessels → wavy.
- Retinal tear .
- Retinal hge → may be seen.



Complications

1- Iridocyclytis
2- Complicated cataract
3- Atrophia bulbi: in neglected cases

↓
Atrophic eye

D.D.

1- Simple from malignant RD :

	Simple (1 ry)	Malignant RD (due to Choroidal malig. Melanoma)
Incidence	More common	Rare
Age	Any	Above 40
History	Trauma (may be)	-ve
Refraction	Myopia (usually)	Any refraction
IOP	Soft	May be ↑
Fundus :		
• **Tear :**	• Present	• Absent
• **Retina :**	• Wavy	• Stretched (tent like)
• **B. V. :**	• Wavy	• Abnormal vascularisation
Investigations :		
• **Ultra-sonography :**	• No mass	• Mass is present
• **Trans-illumination**	• Translucent	• Opaque
• **RadioactiveP^{32} uptake**	• Normal uptake	• ↑ Uptake

Treatment

• Prophylaxis:

- Indications:

1- Flat retinal breaks (without RD)

2- Peripheral retinal lesion predisposing to RD, especially in :

liquefied vit. ← a) Patient: -myopic

– aphakic

– R.D in others eye → Bad history

b) Break: -large – superior → spread rapidly by gravity.

- Technique:

Most breaks are adequately treated by:

* **Laser:** especially if break is large or central (internal ##)

* **Cryotherapy:** especially if (Hazy media or peripheral) (external ##) → because lazer light can't pass

• Curative: (Approximation of retina $\xrightarrow{\text{then}}$ Sealing of break)

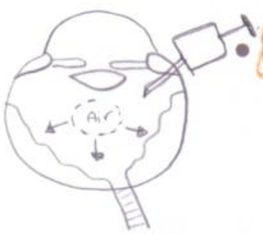
a) The pt. is examined under atropine:

- To determine extent of RD.
- To detect number & site of breaks in 1 ry RD

b) Aim:

- Sealing of the break: by - **Cryotherapy**

  which will induce sterile chorio – retinitis → that heal by chorio – retinal scar → (adhesion between retina & choroid).



(تحزيم ال sclera)

- **Scleral buckling** (pushing the sclera by silicone sponge) → حزام حول ال sclera

  Which help in - Approximation of retina & choroid.

  - Closure of retinal break.
  - Reduction of vitreo-retinal traction

- ± Evacuate the Subretinal fluid (SRF) → only in cases of large fluid
- ± Intra-vitreal injection of air. لأن الأساس هو sealing of break
- **Pars Plana Vitrectomy : in**
  1-Posterior retinal breaks.
  2-Giant retinal breaks.
  3-RD associated with PVR.
  4-To clear vit. Opacities e.g. Hge, to visualise retinal breaks.

(B) 2 ry Retinal detachment

1- Tractional RD :

- in which retina is pulled by vitreous fibrosis

- As in:

✓ -Cyclitic membrane , organized vitreous he,

✓- Proliferative diabetic retinopathy ,

✓- Retinopathy of prematurity,

- Proliferative sickle retinopathy,

- Penetrating posterior segment trauma.

2- Exudative RD:

- in which the retina is pushed by fluid derived from the choroid :

as in ✓ choroiditis as in Harada disease

✓- Neoplasm (malignant melanoma) (M.M. of choroid)

✓- Subretinal neovascularisation

✓- Systemic causes : toxemia of pregnancy,severe hypertension.

TTT : 2 ry RD

Ttt of the cause

1- Tractional: Vitrectomy.

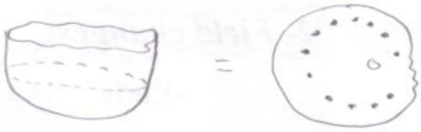
2- Exudative:

- Inflammatory → cortisone. - Malignant → enucleation.

Degeneration of the retina

Retinitis Pigmentosa

= Pigmentary Retinopathy



Definition:

It is familial, bilateral, progressive retinal degeneration of unknown etiology.

ischemia   vit A def.   U.V light
May be due to

Bone Corpuscle like = spider like =

Etiology

unknown , may be

1- Abiotrophy
2- Phototoxicity
3- Hereditary : mode of inheritance may be ,
    - Autosomal dominant
    - Autosomal recessive
    - X-linked النوع الأسوأ

Pathology:

- Degeneration affects the photoreceptors(particularly the rods) &RPE.

-It starts at equatorial region(less blood supply) & progress centrally & peripherally leading to complete blindness at middle age.

-RPE proliferate & migrate inwards toward the inner retinal layers,& is arranged along the small arterioles & venules (bone corpuscles appearance)

working as macrophages to engulf the destroyed rods.

Clinical Picture

➢ Symptoms:

- Night blindness (defective dark adaptation) → Due to destruction of rods.
- then → Progressive visual field contraction.
- then → Finally, Complete loss of vision.

➢ Signs:

1- Fundus Picture: Oral (very imp.)

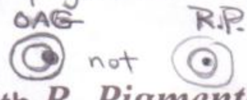
- Retina: Spider like pigmented spots at equator.
- Vessels: Markedly attenuated.
- Disc: Consecutive optic atrophy (Waxy disc).

2- Field changes:

-Early: ring scotoma (due to equatorial degeneration).

- late: tubular field.

Oral: How to defferentiate from ringscotoma of OAG?
1 Tension.
2 Distribution of pigments.
3 Site of blind spot



N.B

Common associations (rather than complications) with *R. Pigmentosa*

1- Local : (i) **OAG** (ii) **Myopia** (iii) **Keratoconus**
(vi) **PVD** (v) **Posterior subcapsular cataract** KOMPP

(Posterior vitreous detachment)

2- General : Syndromes as :

(a) **Bardet- Biedel syndrome : with**
Obesity -Hypogonadism -MR -Polydactyly

لا أسمع، لا أرى، لا أتكلم، دماغه ضاربة (b) **Refsum,s Syndrome : with**
Deafness -Ataxia -Polyneuropathy -EOG changes

Prognosis:

Loss of vision may be due to

1- Complicated cataract.
2- Optic atrophy (consecutive).
3- Macular affection Myopia – glaucoma.

D.D.

1- ***From other causes of night blindness.***
2- ***From other causes of ring scotoma & tubular field.***
OAG

ttt

Useless : 1- Vitamin A. 2- Vasodilators.

The Vitreous

Anatomy

Gross anatomy

- ***Site:*** Posterior cavity between the lens & the retina.
- ***Transparent & clear*** (avascular & contains no nerves).
- ***Surfaces:***
  1- **Outer :** Adherent to the retina at the ora serrata (vit. Base) & around optic disc.
  2- **Anterior :** adherent to the lens by hyalo-capsular ligament "ring like attachment " with retrolental space in-between.
- ***Hyaloid "Cloquet" canal :***

Narrow canal passing from the optic disc, through the central part of the vitreous substance, to the central part of the post. Lens capsule (Canal of Hyaloid artery).

Nutrition

From aqueous, Choroidal & Retina vessels.

Composition

- 99% water.

- The rest is composed of collagen & hyaluronic acid.

Function

1- Optical function : Refractive medium.

2- Maintains the form of the eye.

3- Support for lens & cushion to the retina.

Vitreous Hemorrhage

Causes

➢ ***Local causes :***

1- Trauma (+++)
2- Retinal tear (± R.D)
3- Diabetic retinopathy
4- CRV thrombosis
5- Intra – ocular tumors
6- Eales disease (Periphlebitis retinae of unknown cause may be T.B.)

➢ ***General causes :***

Blood diseases : as leukemia – purpura

Clinical causes :

➢ ***Symptoms :*** rapid ↓ of V.A.

➢ ***Signs :***

- **Fundus exam** → shows the Hge
- **Red reflex** → black

Fate

1- Absorption : slow as the vitreous is avascular.

2- Organization : that may pull on the retina → RD.

Treatment

➢ ***ttt of the cause***

➢ ***Medical ttt :***

1- Vit C (to prevent the recurrence).
2- Alpha – chymotrypsin (to inhance the absorption).

➢ ***Vitrectomy :*** **If organized (for fear of R.D.)**

Musca Volitans

Definition

It is the appearance of moving opacities in front of the eye due to the presence of vitreous Opacities that cast a shadow on the retina.

Causes :

- ***Congenital :*** due to remnants of the hyaloid artery.
- ***Acquired :***
  - **i) Endogenous :** due to changes in the vitreous itself.
    - Protein coagula : when the vitreous becomes liquefied as in:
      1. High myopia
      2. Following trauma
      3. Old age (syneresis)
    - Synchesis scintillans : Cholesterol crystals in the vitreous.
    - Astroid bodies : calcium soaps in the vitreous.
  - **ii) Exogenous :** due to changes in the surrounding structures
    - Inflammatory cells : as in cyclitis : papillitis.
    - Blood cells : as in vitreous Hge.
    - Tumor cells : as in Retinoblastoma.

Treatment

- **ttt of the cause**
- **Vitrectomy :** in some cases

Indications of vitrectomy

(Diagnostic & Therapeutic)

1- Removal of pathological vitreous :

- Old standing vitreous Hge.
- Infected vitreous (Endophthalmitis)
- Tractional RD (vitreous fibrosis).

2- Removal of I.O.F.B. : in the posterior segment of the eye.

Diseases of the optic nerve

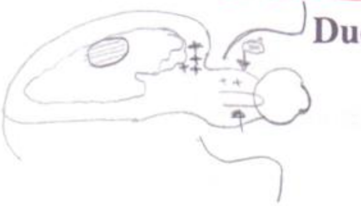
Papilledema

Definition

It is passive (non – inflammatory) edema of the op. Disc.

Active	Passive
Exudate	transudate
Ig ← Protein++/cells	water +++/fibrin
inflammatory	Non-inflammatory
injection	Congestion

Etiology



➢ ***Intra – cranial***

Due to ↑ IC pressure e.g. in

- Neoplasms (70%)
- Inflammation (as in Br. Abscess, meningitis)
- Vascular : subarachnoid Hge , cav. s. thrombosis.
- Pseudo-tumor cerebri (benign ↑ ICT with no IC mass).

➢ ***Orbital :***

Due to e.g. :

- Neoplasms
- Inflammation (orbital cellulitis)

➢ ***Ocular***

Due to e.g.

- CRVO
- Hypotony (V.D → transudation)

➢ ***Systemic***

Due to e.g.

(in old pts)

- Vas. Retinopathy :Malignant hypertension, Eclampsia
- Polycythemia, Anemia.

↓ Polycythemia → ↑ viscosity → Th. in vein

↓ Anemia → ↓O2 → VD of vessels

N.B.

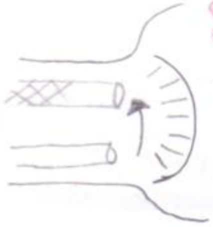
➢ The term ***papillodema*** is usually used to describe disc edema due ↑ ICT , while other causes are usually termed ***Disc edema***

➢ ***Side :***

1- **Bilat. Papilledema :** in I. Cranial & Systemic causes

2- **Unilat. Papilledema :** in Ocular &Orbital causes

➢ *Mechanism (of Papilledema with ↑ ICT)*



1. Mechanical theory Intra – cranial subarachnoid space is communicating with subarachnoid space around op. Nerve. So, any ↑ in ICT will be transmitted to Op. N. pressing on CRV → Transudation.

2- Recently Papilledema is due to block of the axoplasmic flow due to ↑ICT in the subarachnoid space around the op. N. later : the swollen axons compresses the capillaries & veins → congestion → transudation.

Clinical picture

Symptoms

Projectile (not preceeded by nausea)

➢ **Symptoms of ↑ ICT :** Headache – Blurring – Vomiting –Diplopia

➢ **Visual symptoms :**

(i) Early → assymptomatic (blind escaping)
→ (sometimes amaurosis Fugax: transient loss of vision. due to spasm of Cerebral retinal vessels
(ii) Late → gradual progressive Painless diminution of vision (due to optic atrophy)

Signs :

1- Pupil :

a) Early : Normal (RRR)
b) Late : irreactive due to op. n. Damage

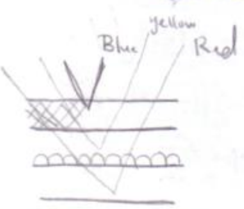
2- Fundus As CRVO

- **Disc** - Hyperemic - ill – defined edges (due to dilated vessels with surrounding choroid)

- Filling of cup (by swollen fibers) - Elevated (up to 9 D= 3 mm)

- **Retinal** *:* flame shaped Hge &Edema
- **Macula** *:* exudate (macular fan)
- **Veins** *:* Engorged & Tortuous

3- Field :



1- Enlargement of the blind spot
2-Central scotoma for blue. (due to its collection in part of oedema)
3-Later : Contraction of field due to op. atrophy.

Normal people

① Myopes for blue
② Normal for yellow
③ Hypermetropes for red

Investigation :

1- Field of vision.
2- Color vision : Central scotoma for blue.
for tumours ← 3- Radiology : a- X- ray b- CT scan
4- VEP : early : normal . Late : affected .

Complications

Post – papilledemic (2ry) Optic atrophy

D.D. Oral هام جداً جداً

صفحة 83

	Papilledema	Papillitis	Pseudo papillitis
I.C.T	Present	Absent	Absent
Vision (هام)	Normal (early)	Markedly ↓	Hypermetrope
Side	Commonly Bilat.	Commonly Unilat	Unilat or Bilat
Field	Central scotoma for blue	Central scotoma for red & green [illegible]	Normal
Fundus	Disc is elevated Up to + 9 D. = 3MM (لأن الماء يخرج بسهولة)	Less elevation (about + 3D)	Less (about +2 D)
Vitreous	Clear	Turbid (inflam. Cells)	Clear
Pupil	Reactive (early)	Reactive (unsustained)	Reactive

Treatment

1- *ttt of the cause .*

2- *Optic nerve decompression operation* (in long standing cases)

Optic neuritis

Definition It is inflammation of the op. nerve.

Classification It is classified into :

1- Papillitis : Inflammation of the disc

2- Retrobulbar neuritis : Inflammation of nerve behind globe. It may be :

- Acute
- Chronic (toxic amblyopia)

Papillitis

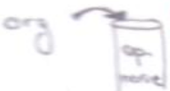
Etiology

1- Antigen -Antibody reaction to a septic focus.

الأشهر والأسوأ **2- Demyelinating diseases especially Multiple sclerosis ~~sclerosis.~~**

Normally no myelin in cornea & opt. nerve fibers (transparency)

3- Metabolic diseases e.g. DM , vitamin B_1 deficiency.



4- Infection :

- 1ry : Viral (polio – Herpes) Bacterial (TB & $)
- 2ry : Extension of infection from
  - a) Brain (meningitis)
  - b) Orbit (cellulitis)
  - c) Ocular (Uveitis & Retinitis)
  - d) Sinuses: Sinsusitis.

Clinical picture

Symptoms : rapid & marked↓ Vision Down to HM. (see table)

Signs :

- **➢ Fundus : ** See table
- **➢ Field : ** See table
- **➢ Pupil** → unsustained reaction
- **➢ VEP** : long latency & diminished amplitude.

D.D. see the table.

Fate

(i) Recovery (in most cases) But recurrence is common.

(ii) Post-papillitic (2ry) Optic atrophy

Treatment

① ttt of cause +

② Cortisone + vitamin B complex + vasodilator.

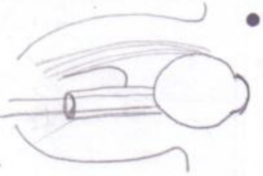
systemic

لأنه بيوصل

Retrobulbar Neuritis

(I) Acute

- **Etiology** : as papillitis
- **Clinical Picture** (*symptoms & signs) :*
  - **Symptoms:** as papillitis
    + **Painful eye movement especially up**
    (as S.R muscle takes insertion from op. n. sheath)



Elevation of inflamed meninges → Pain (when eye elevation by sup. rectus)

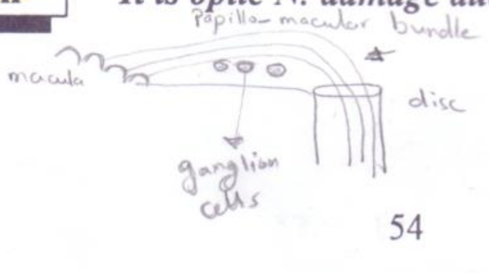
  - **Signs** : as papillitis
    But the **fundus :** is Normal
- **Fate & ttt** : as papillitis

(II) Chronic

Toxic Amblyopia

Oral

Definition ***It is optic N. damage due to "Exogenous toxins"***



Classification

- ➢ *Toxins that produce central scotoma as Tobacco & ethyl alcohol*
- ➢ *Toxins that produce contracted field as quinine & salicylate*
- ➢ *Toxins, that produce severe optic atrophy as methyl alc. & arsenic.*

(1)Tobacco amblyopia :

➢ It is due to degeneration of the ganglion cells of the Papillomacular bundle by the decomposition products of nicotine, either by :

1- Chewing tobacco for many years.
2- Smoking : Cigar & pipes (may be) ,cigarettes (rare).
3- Exposure to tobacco dust in tobacco factories عمال مصانع السجائر

➢ ***Cl. Picture :***

① **fundus** : Normal
② **Field** : Centro – Caecal scotoma (Central to optic disc)

Caecum = blind spot

(2)Quinine amblyopia :

➢ It is due to idiosyncrasy to the drug. في حالات نخس جداً

➢ ***Cl. Picture :***

① **Fundus** : Cherry red spot (due to arterial spasm)
② **Field** : Tubular field (leading to night blindness)

(3)Methyl alcohol amblyopia : (Written)

شرب الخمر المغشوش

➢ It is due methyl alcohol intake or inhalation of its fumes

➢ Pathogenesis : break down of methyl Alcohol in the body → formaldehyde + formic acid → acidosis → Anoxia → - Degeneration of the ganglion cells

➢ *Cl. Picture :*

① Acute stage : Nausea , Vomiting, headache ,Coma & death.
② If the patient survives, vision is usully lost due to Optic atrophy

Treatment

- Stop the drug
- Vasodilators
- Vitamin B complex.
- ***In methyl alcohol Amblyopia***

  1- Oral& IV sodium bicarbonate (for the acidosis) maybe vision & life threatening → to correct acidosis

  2- stomach lavage with ethyl alcohol is required.

Optic Atrophy

Definition

It is a term applied to the condition of the disc when → op. nerve fibers are degenerated due to interruption of the nerve fibers at any point between the ganglion cell layer in the retina & the lateral Geniculate body.

Etiology (types)

1) 1ry Optic Atrophy :

Multiple sclerosis

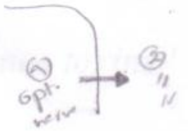
([illegible])

- M. S. (++++)
- Tabes dorsalis ($)
- Tumors : pressing on optic nerve.
- Trauma (fracture base of skull) pressing on op. nerve
- Severe blood loss.(redistribution of blood to vital organs is not to optic nerve)

2) 2ry Optic Atrophy :

causes

(1) Post – papilledemic (2) Post – Papillitic.



① Retina

② optic nerve

(3) Consecutive Optic Atrophy :

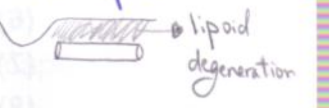
Causes : Following a retinal disease

- CRAO
- R. Pigmentosa
- Degenerative Myopia .
- am. Family Idiocy. عمى عائلي عبيط → Tay Sack's disease

→ lipoid degeneration of ganglion cells

→ Common in Jewish children

→ Fundus picture: Cherry red spot



(4) Glaucomatous Optic Atrophy .

Clinical pictures

Symptoms : Diminution of vision (down to no PL)

Signs :

① **Pupil :** (I) Unilateral cases : afferent papillary defect.

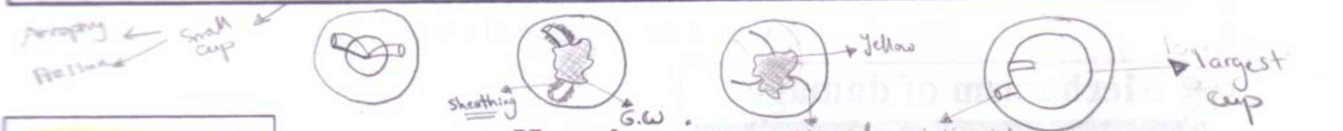
(ii) Bilateral cases : Bilat. Dilated fixed pupil .

② **fundus :** (Oral T.P.(10))

Preceded by edema in → Papilledema / → Papillitis ⇝ fibrosis.(Greyish white)

→ Glial tissue to engulf dead cells (yellow)

	1 ry	2 ry	Consecutive	glaucomatous
(I) Disc				
(1) Color (2) Edge (3) Cup (4) Lamina (5) Vessels	Milky while Well defined Enlarged Seen Normal	Grey white Irregular Obliterated Not seen Sheathed	Yellow (waxy) Irregular Obliterated Not seen Attenuated	Pale white Defined Deep &large (+++) Seen as if Interrupted
(II) Rest of Retina	Normal	Normal	Shows the cause	Normal



Treatment

Hopeless غالبا

ده لو كان المريض لسه شايف

1- ttt of cause

2- vasodilators + vitamin B complex

Foster – Kennedy syndrome

(لازم يكتب الي تحت كاملاً) (Written)

- It's Epsilateral optic atrophy + contralateral Papilledema
- It is commonly seen with frontal lobe tumors

→ 1ry optic nerve atrophy

frontal tumour

↓

No space for edema

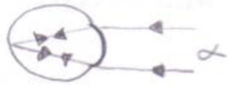
↓

Pressure → Atrophy

Errors of refraction

INTRODUCTION

Emmetropia

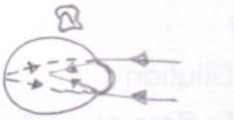


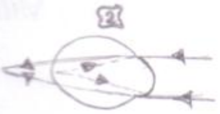
A condition of refraction in which with "Accomodation Completely relaxed" parallel rays come to focus on the retina. Rays coming out from the retina Leave the eye parallel i.e. they meet at لا نهاية infinity.the retina & infinity are called **(Conjugate Foci).** or Reciprocal foci

Ammetropia

A condition of refraction in which with Accomodation completely relaxed parllel rays do"Not" come to focus on the retina.

It includes:
- ① Myopia. ② Hypermetropia.
- Astigmatism.
- Aphakia and Anisometropia.





Factors affecting the eye as an optical system

1- Axial length:

It is the antero-posterior axis normally ≃ 24 mm.

2- Refractive power: depends on

* Curvature of - cornea - lens

* Refractive index (RI).

Accomodation

Definition: It with the ability of lens to change its refractive power so that It can focus object at different distances from the eye.

Mechanism:

Contraction of ciliary ms → smaller ciliary ring → relaxation of zonules → increase curvature of lens (elastic) → increase its power.

Range of accomedation

It is the distance between near point and for pant.

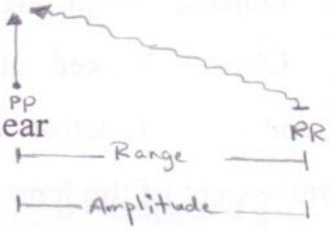
Range: الفرق كمسافة
Amplitude: power الفرق ك

N.B.

1- **Near point** (Panctum proximum)

It is nearest point from eye where objects appear clear

(Here : accomodation is at its maximum).



It depends on age:
5 years: +15 D
35 years: +9 D
40 years: +5 D
50 years: +2 D
>70 years: −D

2- **Far point** (Punctum remotum)

It is most distant point from eye where objects appear clear

(Here : accommodation is completely relaxed).

For emmetrope → at infinity.

Myope → near than infinity.

Hypermetrope → point behind the eye.

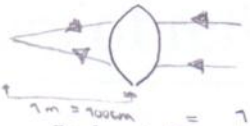
Definition أكثر لأن ليس كل الشروط مستوفاة

Amplitude of accommodation

It is difference between accom for near and accom. for far

= refractive power at near – refractive power for far

The Dioptre



∴ 50 cm 2D
∴ 25 cm 4D

It is the unit of lens power. It is defined as the power of a lens which brings parallel rays falling on it to a focus at a distance of one meter.

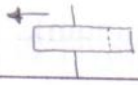
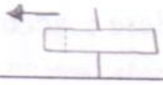
Types of lenses

جزء من كرة زجاجية

1- Spherical lenses

- Are segments of spheres, brings light rays → point of focus.
- 2 Kinds : concave and convex.

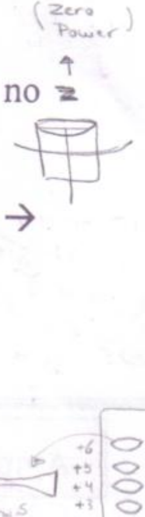
(Oral مش مهم)

Convex	Concave
Thick at centre-> periphery	Thicker at the periphery.
Objects appear larger	Objects appear smaller.
Objects looked at move in opposite direction to the movement of the lens 	Move in the same direction 

جزء من اسطوانة زجاجية

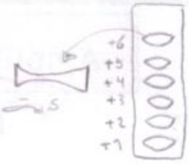
2- Cylindrical lenses

- Are segments of a cylinder.
- Light rays passing in a plane of axis of the cylinder → undergo no refraction.
- Light rays passing through the cylinder perpendicular to its axis → undergo xaximum refraction.



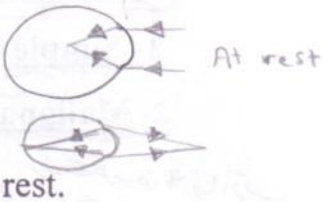
Estimation of the lens power

1- By neurtilization with lenses of opposite kind and known power.
2- Phacometer (lens meter) = focimeter.



صندوق
عدسات
محدبة
(وأجربهم واحدة واحدة)

Myopia



Definition

It is a condition of refraction in which with accomodation at rest.

- Incident parallel rays come to focus in a "Point" infront of retina.
- Rays emerging from a point on retina. Leave eye convergent and meet at a point in front of the eye (punctum remotum).

Aetiology

1- Axial myopia (commoest type)

Due to increase in axial length of the eye. It may be :

(Types of axial myopia)

	1- Simple	**2- Degenerative (Malignant)**	**3- Congenital**
- Onset.	* Around 14 ys(puberty)	* Around 7 ys(على سن المدرسة)	* Since birth
- Progress till.	* About age of 25 ys	* Continue after 25 ys	* Stationary → نظره لا يضعف مع الوقت
- Degree.	* Less than –6D	* may reach > -25D	* About –10D
-Degenerative changes	* Absent	* Present	* Less

2- Refractive mayopia

Due to increased refractive power of eye it includes.

Curvature myopia: as in a: Increae curvature of cornea (keratoconus)

b: Increase curvature of lens (lenticonus)

Index myopia: a- Increase R.I of nucleus (Nuc. Cataract)

b- Decrease R.I of cortex (uncontrolled D.M).

Clinical picture

* Symptoms

1- Simple → indistinct far vision. but can accommodate for near vision

2- Malignant: i- Indistinct far vision.

إرهاق العين

ii- Discomfort after near work (Muscular athenopia).

أكثر راحة للمريض لأنها أقرب إلى Retina من meeting point of far vision

iii- Musca volitans: due to vitreous degeneration. (if fibers of vitreous are cut)

iv- Photopsia: due to stimulation of rods &cones by V-R traction

v- Night blindness: due to degeneration of retinal periphery.

due to ischemia by stretch of vessels & peripheral ones are more affected as they are smaller → so, rods are early affected

(The myopic patients usually screw their lids to simulate a pinhole)

*Signs

1- Simple → normal Eye.

2- Malignant:

i- Oblique illumination : - Large eye: -Large cornea -Deep A.C.

- Pseudoproptosis - bule sclera

ii- Fundus examination: Tigroid fundus (due decreased ret. Pigmentation).

iii- Retinoscopy

Complications

Written & Oral very imp.

1- Complicated cataract.

2- Squint: Mild error : بيروح و بيجي - Latent divergent squint (exophoria)

High error : موجودة على طول - Manifest divegent squint (exotropia)

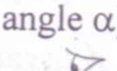
- Apparent convergent sq.(due to -ve angle α)

-- acc → -- Conversion

→ MR<LR

↓

بسبب تعود الشخص على عدم استخدام accommodation مما يؤدي إلى تقوية lateral rectus على حساب medial rectus

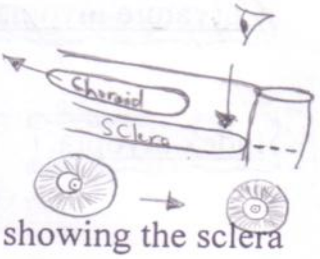


3- Retinal changes

i- Temporal crescent : - It is a white cresent

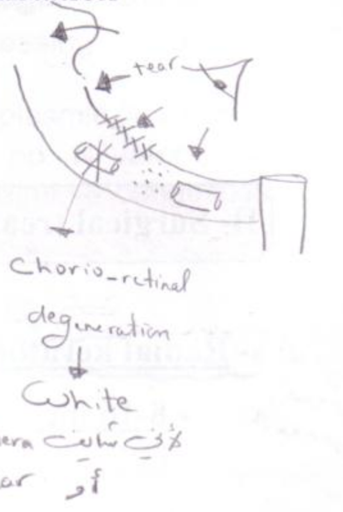
- Site: temporal to disc

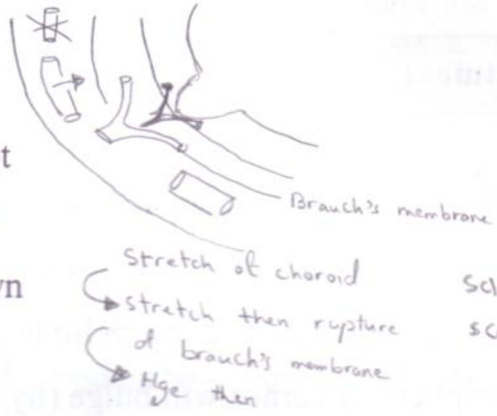
- Cause: traction on the choroid showing the sclera



ii- Chorio-retinal degeneration,scars & atrophy

- It is white areas surrounded by pigmenetation
- Site: peripheral (+++)
- It may → tear → R.D.





iii- Fuch's spot:

- It is a dark spot
- Site: at macula
- Cause: unknown

→ Due to fibrovascular invasion of fovea after rupture of bruch's membrane.

4- Vitreous degneration

The vitreous becomes - Liquified

- Contains opacities (musca volitans) → Coiled fibers

5- Posterior staphyloma

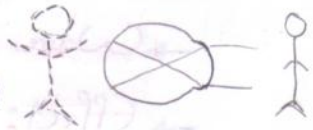
- It is ectasia of posterior segement
- Site: temporal to disc.
- Seen: by fundus examination.

Treatment

I- Optical treatment

i- **Glasses** (concave or minus lenses).

- **Simple myopia**: give full correction (least power → 6/6)



- **High myopia:**

Children: give full correction (to allow normal mental dev)

عايز يعمل نظارة أول مرة Adult: give him undercorrection (why?)

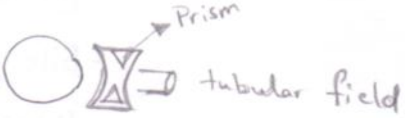
- Because the patient is not accustomed (متعود على) to the small, sharp and bright retinal image given by the high minus lens → ocular discomfort.

  (So, for a myope –20.0 D give him e.g. - 18.0D).

  المريض مش متعود على الضوء العالي ده

ii- Contact lenses

Advantages
- 1- Cosmetically better.
- 2- Bigger field. not →



- 3- No significant decrease in retinal image size.

II- Surgical treatment (refractive surgery)

i- Radial keratotomy (rarely used nowadays)

تشريط القرنية

الهدف هو إضعاف الcornea

- 8-16 incisions (deep and radial) are made from the edge of the optical zone → limbus.
- Idea: the periphery of cornea will bulge (by the IOP) and centre flatten. → ↓power → Collection of light on retina
- Limits:
  - Degree: not less than –2D and not more than –4D (simple myopia)
  - Age: not before the age of 20 ys → to ensure stable myopia.

خوفاً من overcorrection or undercorrection

ii- Excimer laser: to flatten the central cornea.

Technique : either by 1- PRK. 2- LASIK

iii- Phakic IOL.

لأنه أساساً عنده lens

iv- Clear lens extraction.

v- Scleral Resection :was tried for high myopia with no success

كرة محترمة

Excimer laser (مهم جداً)

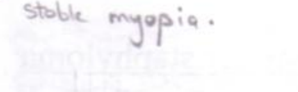
→ Excited dimer

→ Wave length = 193 nm (UV rays)

→ Effect: Photoblation (الاستئصالي)

→ Techniques

① PRK (Photorefractive Keratotomy):



- Disadvantages → Ⓐ Severe postoperative pain (by corneal ulcer)
  Ⓑ Postoperative haze (Bowman memb. damage)

② LASIK (Laser insitu Keratomeleusis):

→ Excimer laser is applied to exposed stromal bed after elevating corneal flap

Advantages: → Ⓐ Less pain Ⓑ Less haze Ⓒ More predictable results.

Hypermetropia

Definition

It is a condition of refraction in which with accommodation at rest

i- Incident parallel rays come to focus in a point behind the retina.

ii- Rays emerging from a point on the retina leave the eye divergent and appear as if coming from a virtual point behind the retina (p. remotum).

Aetiology

عين حجمها صغير

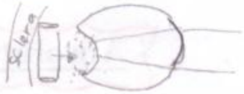
1- Axial hypermetropia

Due to small antero-posterior axis (small eyes) seen in children (below the age of 7 ys) as the eyes are small. As the eyes grow, they become less hypermetropic:

- If the eyes reach the proper length (24 mm) → emmetrope.
- If the eyes don't reach the proper length → remain hyper.
- If the eyes exceed the proper length → become myopic.

(It may be acquired , if the retina is pushed forward as in CS retinopathy).

↓ Central serous



stress → VD of choroid vessels → exudate pushes retina forward.

2- Refractive hypermetropia

Due to decrease refractive power of the eye.

i- Curvature H.: due to flattening of the cornea (cornea plana).

ii- Index H.: due to increase RI of the lens cortex (Imm. Cortical cat).

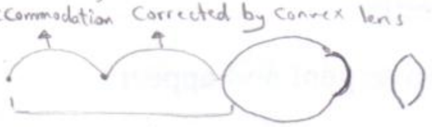
3- Aphakia and posterior lens dislocation.

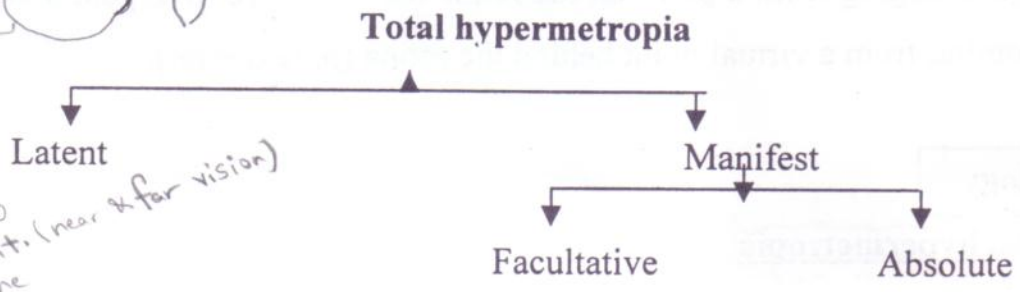
Oral: When manifest = facultative hypermetropia?
- Children.
- Very low errors.

Components of hypermetropia

Oral very imp.

In a hypermetropic patient, the tone of the cliary ms corrects a portion of hypermetropia (latent H.) and is about 1 dioptre. The remaining portion of hypermetropia (Manifest H.) may be corrected by accommodation (facultative H.). If the patient cannot correct all his manifest hyp., a part will remain uncorrected (absolute H.) and leads to decrease of vision.





ابدأ تخيل Hypermet. يصلح بالـ accommodation وإذا ciliary ms اشتغل كتير بيحصل continued cont. → headache

- **Total H.:** Amount of H. measured under the effect of atropine (= the power of convex lens →6/6 (with atropine).

الكامنة

- **Latent H.:** Amount of H. corrected by the tone of ciliary ms. (= total – Manifest).
- **Manifest H.:** Amount of H. not corrected by the tone of ciliary ms Def: (= highest power convex lens → 6/6 (without atropine).
- **Facultative H.:** the part of manifest H. corrected with accommodations Def: (= manifest - absoulte).
- **Absolute H.:** The remaining part of manifest H. not corrected by accom. (= least power convex lens → 6/6 (without atropine).

Clinical picture

*** Symptoms**

a- Young age: no symptoms (accom. is strong and correct all H. i.e.M=F
+15 D

b- With advance of age: the pt. Finds difficulty especially in near vision:
+9 D

- Early-presbyopia
- Accommodative athenopia (يسبب إجهاد العين due to Continued Ciliary cont.)

c- Old age: difficulty in far 8 near vision.
0 D

لا شايف بعيد و لا شايف قريب

*** Signs**

1- Mild degree: as normal eye (مرض أوي أكسانة)

2- High degree:

- Oblique illum.: small eye (Small cornea- Shallow AC- Small pupil).
- Fundus exam.: - Bright reflex- Tortous retinal vessels- Pseudopapillitis
- Retinoscopy

* Pseudopapillitis: ill-defined slightly elevated optic disc due to crowdening of retinal nerve fibres in a small lamina cribrosa.

++ acc → ++ Conversion
→ MR > LR

Complications

1- Squint: - Mild degree: (بيروح بيجي) - Latent convergent squint(esophoria). (inside latent)

- High degree: (على طول) Manifest convergent squint(esotropia).

- Apparent divergent squint.

2- Angle closure glaucoma

Treatment

*** Mild degree:** No symptoms → so, no ttt.

*** High degree:**

1-Optical correction - Glasses (convex lens) Or - Contact lens (C.L → من دروس Myopia (لازم تكسب مهارات

i- Children: give full correction

ii- Adult * 1st give the highest "tolerated" convex lens (as the patient cannot tolerate full correction because he cannot relax the ciliary ms at one time due to its spasm. * After 6 months → give full correction.

iii- Old: for far → give full correction.

For near → give full correction + 3 diopters → Because average reading is on about 30 cm.

2- Surgical ttt: 1- Lasik 2- Holmium Laser thermokeratoplasty

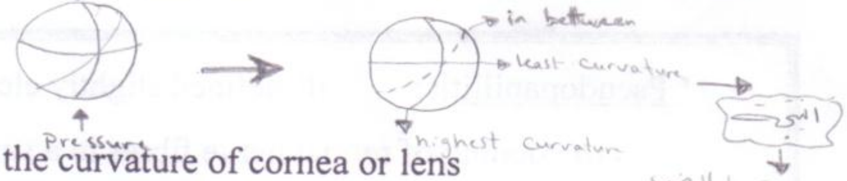
→ 8-16 periphral laser shots, leading to shrinkage of stromal collagen & causing increased curvature of central cornea.

Stigma = one point

Astigmatism

Definition

It is a condition of refraction in which the incident parallel raye don't come to a point focus on the retina (as the eye doesn't have the same power in all meridia).



عند سقوط الضوء لا يتجمع في نقطة واحدة لاختلاف Curvatures

Aetiology

Due to irregularities in the curvature of cornea or lens

1- **Corneal astigmatism:** most of cases, as in { Keratoconus / Opacities

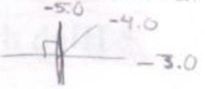
2- **Lenticular astigmatism:** as { Lenticonus / Subluxation

Types

I- Regular

According to axial length

- Menidia of highest and least powers are perpendicular to each other.
- Transmition from the highest meridian to the least is gradual. → oblique meridian is inbetween



* It includes:

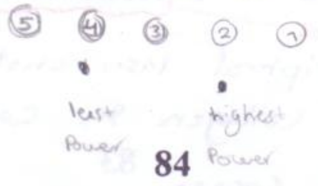
1- **Simple astigmatism:** in which { one meridian → emmetrope / the other ametrope

so, we have ④ i- simple myopic ast. (all rays in front of Retina) ③ ii- Simple hyperm. Ast. (all rays behind Retina)

2- **Compound Ast.:** in which → both meridia are ammetrope but of the same type.

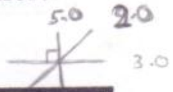
so, we have i- Compound myopic ast ([illegible] Retina [illegible]) ⑤ ii- Compound Hyp. ast. ([illegible] Retina [illegible]) ①

③ 3- **Mixed Ast:** in which → one meridian myopic and the other hypermetropic.



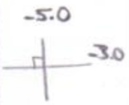
II- Irregular

- Meridia of highest and least powers are "not" perpendicular to each other.
- Transmition from the highest meridian to the least is "not" gradual.



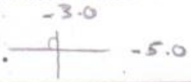
Rule of astigmatism

In emmetropia: the veritical meridian (due to pressure by the lids on the cornea) is more curved than the horizontal → more powerful → mayopic. → less hypermetrupic



In astigmatism: if the vertical merdian is more myopic → ast. "With the rule".

If the horizontal meridian is more myopic → ast. "against the rule".



Clinical picture

*** Symptoms**

- Indistinct vision. (As any error)
- Accommodative athenopia. (في بعض الحالات ← صداع مستمر)
- Distorted objects. (مفيش حد أبداً بيشتكي من الشكوى دي) → Due to difference of meridia

*** Signs:**

حالات نادرة 1- Gross signs: e.g, keratoconus, corneal opacities, subluxation.

حاجات بسيطة

2- Landolt's chart: "some" openings in "the samle line" are not seen. → بيغلط في بعض العلامات رغم إنهم في صف واحد (مش زي Myopes or hypermetropes اللي بيشوفوا الصف كله أو لأ)

3- Placcido's disc: shows irregular circles.

4- Astigmatic fan. (المريض شايف الخطوط في المنتصف بوضوح وممكن على الأطراف بهتانة)

5- Ophthalmoscopy: the optic disc appears oval. → الحاجة الوحيدة اللي round في fundus وأشوفها oval من خلال irregular cornea

حاجات محترمة

6- Keratometer: the curvature (and the power) of cornea can be measured.

7- Retinoscopy by red reflex

8- Autorefractometer : Better

9- Corneal tobography

Treatment

Oral very imp.

*** Optical ttt**

A-Regular ast: corrected by
- * Glasses
  - Cylindrical.
  - Sphero-cylindrical.
- * Contact lenses (soft toric or rigid).

So:

1-Simple ast.: Give cylindrical lenses with its axis perpindicular to amm meridian.

2-Compound or mixed: Give sphero-cylindrical lenses.

B-Irregular ast: → 1-give rigid C.L.

2- PK : if vision doesnot improve with rigid C.L.

No identical refraction

Anisometropia (Refraction of 2eyes not equal)

Definition: It is a significant difference in refractive error between the 2 eyes (rarely tolerable when the difference > 4 diopters).

Aetiology 1- congenital. 2- acquired e.g, unilateral aphakia.

Problem: cannot be corrected by glasses (why?). Anisokonia → Bilateral diplopia

Symptoms : 1- Athenopia. 2- Difficult binocularity 3- Exotropia.

Treatment:

1- Glasses: can be used after undercorrecting the eye with a higher error (at the expence of good vision)

2- contact lens: produce ret. Image difference 6-10 %

3- IOL: reduce the ret. Image to less than 1%.

4- refractive corneal surgery.

Aphakia Written & Oral Ī p. 16

- **Definition**: It means absence of lens
- **Aetiology**:

1- Congenital v. rare.

2- Acquired: i- Surgical removal (most common).

ii- Posterior dislocation of lens.

iii- Trauma to the lens in children (with opened capsule → absorption of lens matter).

- **Clinical picture**

Symptoms: defective vision especially for near (no accommodation).

Signs: (Oral Ī p. 16)

1- +ve history of operation.

2- Scar of previous operation.

3- A.C → deep.

4- Iris - Iridectomy. which done with ICCE for fear of pupillary block - Tremulous (lack of support)

5- Pupil - [illegible] Jet black. (Because light will reflect to source of light not to my eye.

6- Absence of 2 out of 3 purkinge images. (catapteric images)

7- Refraction: shift "toward" hypermetropia.

- **Optical condition after lens extraction**

1- Loss of accommodation.

2- Astigmatism against the rule: about 2 diopters (due to flattening of vertical meridian by scar of operation at upper limbus). but in temporal side → with the rule.

3- Loss of a refractive surface: refraction toward hypermetropia:

a- A previously emmetropic patient → hypermetrope + 10.0.D (فـ نظارة)

b- A previously hyperm. Patient → hypermet. $\underset{>}{\le}$+ 10.0.D

c- A previously myopic patient.

- Simple myopia → hyper. < + 10.0 D
- Degen. Myopia. → much less myopia.
- Theorticaly a myope –25.0 → emmet.

Treatment

1-Glasses:

***Disadventages:** 1- large image (25-30%)

2- aberrations

3- Prismaticeffect

4- limited field

5-can not be used in unilateral aphakia (anisokonia)

2- Contact lenses :

rarely tolerable due to - loss of accommodation&

- difficult dexterity inold age(↓ القدرة على استخدام C.L in old age)

3- IOL Implantation :either 1ry or 2ry

So, **Bitateral aphakia** can be corrected by:

- Glasses. - Contact lenses. - IOL

Unilat: aphakia can be corrected by:

- Contact lenses. - IOL. - But not glasses

Presbyopia

Definition

(Oral (b)

It is ①physiological ②recession of near point (p.p) due decrease in accommodation power making near work ③uncomfortable.

Aetiology

Due to ~~decrease~~ increase lens sclerosis as age advances

→ decrease in its elasticity → decrease in accom power.

Clinical picture

1- Difficulty in near work. (بأتعب لما بأقرأ الجرنان)

2- The pt. Hold the book at a greater distance. (لازم أمسك الجرنان من بعيد علشان أقرأه)

3- Accommodative athenopia → headache. (لما بأقرأ الجرنان مدة طويلة بيجيلي صداع)

N.B the onset of presbyopia varies with the refractive state of eye

- Emmerrope : around age of 45 ys.
- Hypermetrope : before that age.
- Myope : after 45 ys.

Treatment

By reading glosses

Contact lenses

Very thin lenses applied directly to the cornea

Principle

Abolishes the cornea as a refractive surface and replaces it by "contact lens-fluid lens system".

Indications (Oral)

1- **Optical**
- High errors
- Unilat aphakia.
- Irregular astigmatism.

2- **Therepeutics**
- Neuroparalytic keratitis. Resistant ulcers.
- Keratitis metaherpitica.

3- **Cosmetic**.

4- **Occupational**.

Types

1- Hard (PMMA) 2-rigid (silicone) 3- Soft(hydrogel plastics)

Advantages

1- Larger field. No frame + move with the eye

2- No ansiokonia → No binocular diplopia.

3- Cosmetically better.

4- Avoid aberratios which result from prismatic effect of lens periphery.

Disadvantages

1- Intolerance by some (>with hard) & allergy by others esp. with soft C.L.

2- Corneal abrasion or keratitis. → by antigen

3- Lens infection and deposits: if not properly cleaned.

& forign body sensation.

(ملغي)

Retinoscopy (skiascopy)

- **Definition:** it is a method for estimation of refraction of eye.
- **Instruments:** 1- Lamp and plane mirror or electric retinoscope.
  2- Trial frame.
  3- Lenses of different powers.
- **Technique:**

1-Cycloplegia:
- Children: atropine oint 1% rds/3 days estang cling).
- Above 10 ys: - homatropine 1%
  - Mydriaticyl 1%

2- **The patient sits in a dark room** at a distance of one meter from the examiner.

3- **The observer reflect the light into the patient pupil** to see the red reflex. Then, the mirror is moved and the movement of the red reflex is noted.

4- **The movement of the RR may be one out of three:**

- Against (to the opposite direction to that of mirror) → myopia > 1D.
- No movement → myopia –1.0 D.
- With movement (to the same direction as the mirror): may
  - Myopia < -1.0 dioptre.
  - Emmetrope.
  - Hypermetropia.

If RR moves with add plus lenses and if moves against add minus lenses in an ascending fashion till the movement of RR stops → neutral point.

5-**Astigmatism is diagnosed** when one meridian is corrected while the other is still moving. The power of the lens which correct the first meridian is noted and more lenses are used till the second is neutrilized.

6- **Prescription is given to the patient**

add - 1.0 D : for the distance of one meter.
- 1.0 D : if a cycloplegic is used.

The extra-ocular muscles

There are 6 EOM (4 recti and 2 obliques) which rotates the eye,around :

3 axes → (i) **Vertical axis** → adduction and abduction.

(ii) **Horizontal axis** → elevation and depression.

(iii) **Antero-posterior axis** → intorsion and extorsion.

Rectus muscles

- **Origin:** arise from a fibrous ring placed around the op. Foramen and medial part of SOF → annulus of Zinn.
- **Insertion** to the sclera at a variable distances from the limbus.

Oblique muscles

- **Origin:**
- Superior oblique: from the orb. Apex above and medial to the an. of Zinn.
- Inferior obliques: from the orb. Floor lateral to the opening of NLD.

- **Insertion**

S.O: the ms passes forward and medially then become tindinous and pass through a fibrous ring at the artero-medial part of the orbital roof near the orbital margin :trochlea" to be reflected backward and laterally to be inserted in the sclera in the upper postero-lateral quadrant.

IO: the ms passes backward and laterally below the globe to be inserted in the sclera in the lower postero-lateral quadrants (5 mm from op. nerve almost over the macula).

- **Nerve supply**

  - 3rd n. (oculomotor): sup. Rectus, MR., I.R, Inf. Oblique.
  - 4th n. (trochlear): superior oblique.
  - 6th n. (abducent): lateral rectus.

- **Blood supply**

  Muscular branches of ophthalmic artery

- **Action**

  1- Medial rectus: adduction

  2- Lateral rectus: abduction

  3- Superior rectus: elevation (Primary action)
  : adduction & intorsion (subsidary action).

  4- Inferior rectus: depression(Primary action)
  : adduction & extorsion (subsidary action).

  5- Superior oblique: intorsion(Primary action)
  depression & abduction(subsidary action)

  6- Inferior oblique : extorsion(Primary action)
  elevation & abduction(subsidary action).

- Superior muscles are intorters while inferior muscles are extorters.
- Recti are adductors while obliques are abductors.

The muscle axis

It is the line between the origin and insertion

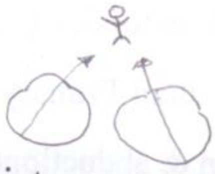
- **The mulsce action is:**
  - → maximum if ms axis is parallel to visual Axis.
  - → nil if ms axis is perpendicular to visual Axis.

Squint

Definition

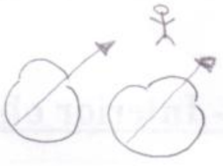
A condition in which the visual axes of the 2 eyes are "not" directed to the same object.

Types



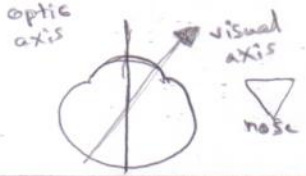
1- **Apparent squint**

2- **True squint:**
  - Latent squint
  - Manifest squint:
    - Paralytic,
    - Concomitant.



N.B.

- **Nodal point:** It is a point behind the posterior pole of the lens through which light rays undergo no refraction.
- **Visual axes:** It is the line connecting the macular and object of regard passing through the nodal point.
- **Optic axis:** It is the line connecting the centre of the cornea, leas, retina.

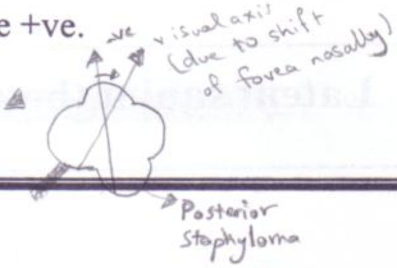


Angle alpha : It is the angle between the visual axis and op axis.

In emmetropes 5° &+ve (v.axes cut the corena nasal to op.axis)

In hypermetropes → It is large +ve.

In high myepia → It is -ve.



Apparent squint

Definition

A condition in which the person appears to have squint although the 2 visual axes are directed to the same object.

Etiology

1- **Apparent convergent squint:**

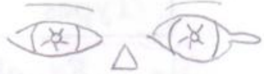
a- Epicanthus: a skin fold over the medial conthus congenital, racial.

b- High myopia: due to –ve angle alpha.

c- Small IPD (normal IPD : 55-70 mm).

2- **Apparent divergent squint:**

a- Lateral ankyloblepharon: adhesion between the hid margins.



b- High hypermetropia: due to large +ve angle alpha .

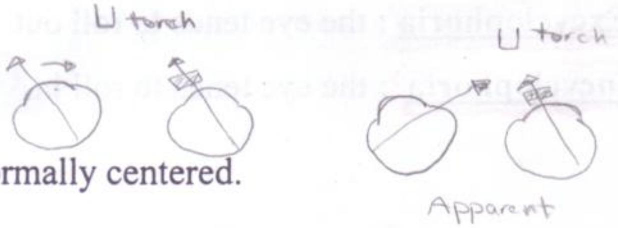
c- Large IPD.

Diagnosis

1- Presence of a cause.

2- Cover test → no movement.

3- Corneal light reflex : may be normally centered.



Treatment:

Treatment of the cause only (→ as there is no squint). → إياك تعمل كده لأنها معدوك ولو دقتها هتعمله (True squint)

في الـ ث من الاهم ده

Latent squint (heteropharia)

Written Imp هام

Definition

Oral Imp هام

A condition in which the eye has a ① tendency to deviate (when the patient is fatigued or the brain loses interst in binocular single vision) due to ② EOM imbalance "but" this tendency is checked by the brain ③ subconciously to maintian "④ binocular vision".

Etiology

EOM imblance

1- **Errors of refraction:** leading to disproportion between accommodation and convergence.

2- **Mild weakness of one or more of EOM** not sufficient to produce → manifest squint.

Types

1- **Exophoria** : the eye tends to deviate outwards.

2- **Esophoria** : the eye tends to deviate inwards.

3- **Hyper phoria** : the eye tends to deviate ~~inwards~~.upwards.

4- **Hypophoira** : the eye tends to deviate downwards.

5- **Excyclophoria** : the eye tends to roll out. (When inferior " " " ")

6- **Incyclophoria** : the eye tends to roll in. (When superior oblique takes upperhand)

Oral

Accomodative athenopia.

① Hypermetropia.
② Astigmatism.
③ Presbyopia.

Symptoms

1- **Compensated cases:** show no symptoms (most cases).

2- **Decompensated cases:** may show.

الي حواليك بياخدوا بالهم لما تسرح أو تتعب

a- Symptoms due to trial to maintain binocular vision → muscular athenopia (eye strain). After prolonged close work, the eyes are red with:

- Ocular pain. – Headache. -Lacrimation. → وكأن العين بتحرق من التعب

انت بتاخد بالك لما السطور بتدخل في بعض.

b- Symptoms due to failure to maintain Binocular vision.

a- Running letters b- Latent → manfiest.

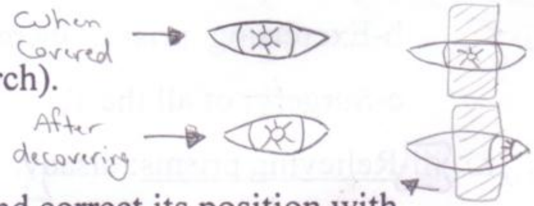
squint latent

Diagnosis

Principle: depends on abolishing the B.V by covering one eye, presenting different images to each eye, so, the brain will lose interest of B.V. and the latent squint will become → manifest squint.

1-Cover-uncover test

- Ask the patient to fix an object (tourch).
- Cover then uncover one eye.
- If this eye deviate under the cover and correct its position with uncovering → latent squint.



2-Maddox rod

أسماء مؤجلة

Maddox rod is a group of red cylinders arranged parallel to each other.
They produce a red line image from a point source of light
(this red line is perpendicular to the axis of the cylinders).

- The rod is placed in front of one eye and the patient fixates a source of light at a distance of 6 meters).
- An orthophoric (normal) sees the point of light coinciding with the middle of the red line.

أسماء مؤجلة

3- Maddox wing

- The Rt. Eye sees 2 arrows (vertical & Horizontal).
- The Lt. Eye sees 2 scales (V. & H.).
- An orthrophoric sees the arrows points to the → zero.
- In Heterophoria (the patient can read directly his angle of squint).

Treatment

لما المريض يكبر

1- **Compensated cases**: no symptoms → No. ttt.

2- **Decompensated cases**.

i- Correct any error of refraction.
ii- Strengthen the weak ms by:

ttt of causes

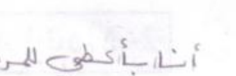
بيطلع قلم قريب من عينه ويشوفه كل يوم ربع ساعة أو نصف ساعة

a-Orthoptic exercises: esp. in convergence insrfficiency.

b-Exercising prisms: increase muscular effort.

المريض هيحس كأنه تعبان: أنا كنت في الأول أذاكر ساعة وأتعب دلوقتي مع النظارة الجديدة أتعب أسرع ← بسبب تمرين العضلة لكن مع الوقت الوضع هيتصلح.

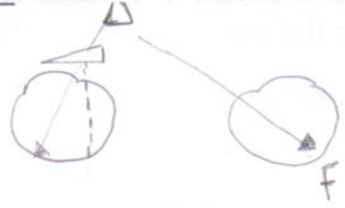
c-Surgery: of all the above failed (in large degrees).



ophthalmic Prism.

iii- Relieving prisms: disadv. → latent → manifest

للناس الكبار



:أنا بأعطي للمريض الاختيار للعلاج بـ ال prisms

← هقعد أعالجك بـ (exercising prism) وهتتعب معايا شوية بس في الآخر منظر عينك هيتصلح (الشباب)

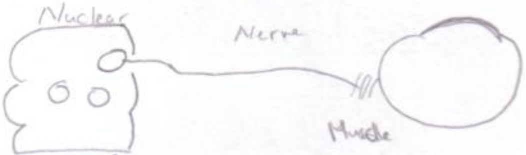
← وإلا انت مش عايز تتعب وأعطيك (relciving prism) يعالج نظرك بس منظر عينك مش هيتصلح (كبار السن)

Paralytic squint

Written هام

Definition

It is manifest squint due to paralysis of one or more of EOM.



Etiology

It is due to lesion in the muscles or its nerve supply.

1- Nuclear lesions

- Congenital : eg. Absence of the nucleus.
- Inflammatory : e.g encephalitis.
- Vascular : e.g, thrombosis, embolism,hge
- Neoplastic : tumors of brain stem (Midbrain& pons).
- Other (toxic) : as in diphtheria and with alcohol, lead.

2- Nerve lesions

- Congenital.
- Inflammatory : meningitis, cav. Sinus thrombosis.
- Vascular : Hge (subdural,subarachnoid)& cav. sinus Thrombosis.. , Microangiopathy (D.M)
- Neoplastic : Brain tumor (ICT),orb. Tumor.
- Trauma : e.g, fracture base of the skull.

3- Muscle lesion

- Congenital : e.g maldevelopment of the ms.
- Trauma : Fracture orbital bones.
- Neuro-muscular:Myopathy & Myathenia gravis. → أهم
- Thyrotoxic exophthalmos.
- Neoplastic : orbital tumors.

4_Supranuclear lesion

Gaze paralysis (2 eyes don't move at same time)

N.B.

***Opthalmoplegia**

1- External opthalmoplegia → paralysis of EOM.
2- Total ophthalmoplegia → paralysis of EOM and intra OM.

***Superior orbital fissure syndrome:** it includes affection of 3rd, 4th, 6th and 1st division of the 5th cranial nerves.

***Orbital apex syndrome.** It includes SOF syndrome op.n affection.

Clinical picture (Oral (١٥)

→ All of them are in the same direction of action of paralyzed ms. except manifest squint

1- **Manifest squint:** to the "opposite" direction of the action of the paralysed ms. (the paralysed ms. Loses its tone → the antagonistic draws the eye toward it)

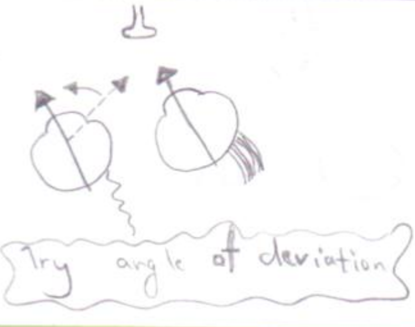
Oral (١٥)

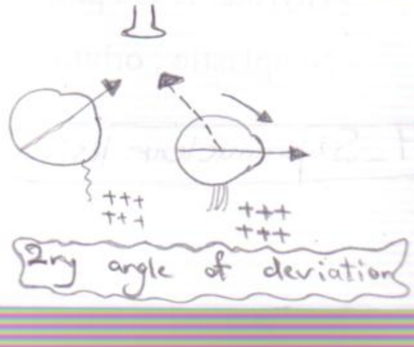
N.B.: The angle of squint: in paralytic squint

The 2ry angle of deviation (when the paralyzed eye is fixing)	>	the 1ry angle of deviation (when the normal eye is fixing).

ii- Explanation:

- Normally, the impulses reaching the 2 eyes in order to look to any direction are equally distributed between the 2 eyes (Hering law).
- When the paralysed eye is fixing (the normal eye is covered) → more impulses reach the paralysed ms to force it to contract. But, at the same time the normal eye (synergistic ms) will receive equal amount of impulses. Thus, the normal eye will move much more (under the cover) → larger angle.


1ry angle of deviation



2ry angle of deviation

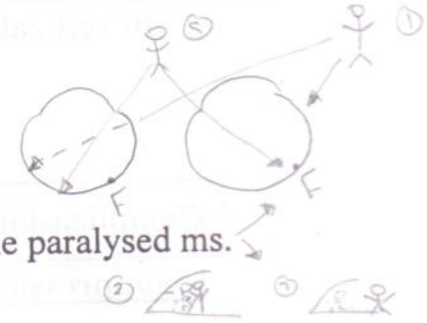
2-Limitation of ocular movement: in the "same" direction of action of the paralysed ms.

3- Diplopia:

Cause: the image falls on non-corresponding retinal points.

- The image seen by the normal eye is clear as it falls on the fovea → true image.
- The image seen by the squinting eye is blurred as it falls out side the forea → false image.

Characters:



① Binocular: disappears if one eye is covered.

② More in the "some" direction of action of the paralysed ms.

③ It may be crossed or uncorossed.

ملغي

~~**N.B.**~~

~~1- **Uncrossed (Homonymos) diplopia** when the false image falls on the same side of the paralysed "eye" e.g, in lat. R. paralysis.~~

~~2- **Crossed (Heteronymos) diplopia:** when the false image falls on the opposite side of the paralysed eye e.g, in med. R. paralysis.~~

The patient fights diplopia by:

- Suppression (child).
- CHP.
- Covering one eye.

4- False project (past pointing)

It is false orientation of objects situated in the "same" direction of action of the paralysed ms i.e wrong estimation of the site (with the normal eye covered).

Cause: the brain sends excessive impulses to the paralysed ms to force it to contract.

5- Headache, Nausia, Vertigo: (transmission from trigeminal to vagus nerve)

iii- Cause : the diplopia and the false projection.

6- Compensatory head posture (ocular torticollis)

- It is abn. Head posture adopted by the patient to minimize diplopia.

- It occurs in the "same direction of action of the paralysed ms.

e.g, In horizontal deviation → simple (face turn).

In vertical deviation → compound:
- face turn. due to abduction & adduction
- chin elev. Or depression.
- head tilt. for intorsion & extorsion.

Complications

i- Suppression: of the false image (to avoid diplopia)→ amblyopia . ex an opsia out No Defective vision

Brain → suppression (transient) → if prolonged كسل

ii- Ms changes:

Direct antagonist overaction due to contructure (on prolonged palsy).

Atrophy → fibrosis → Contracture

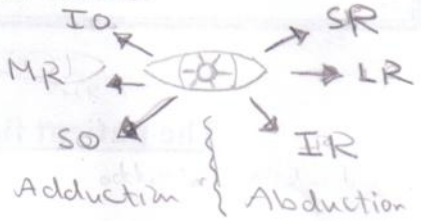
Diagnosis

الأهم

1- **Testing of ocular motility** ask the patient to fix your finger in the 6 cardinal directions.

2- **Diplopia chart.**

3- **Hess screen.**



Treatment

1- Treatment of the cause:

This may lead to complete recovery (spontaneous nerve regeneration).

During that → cover one eye (to avoid diplopia).

نيجي العملية

2- Surgical treatment:

Required if no recovery occurs after 6 months of ttt.

الغاء Diplopia in Primary position → Frozen globes (يعني المريض ميحركش رأسه علشان ميحصلش Diplopia)

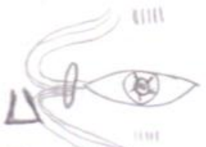
a- If the ms is weak (the paralysed eye cross the mid-line ie.paresis)

→ strengthen it & weaken its direct antagonist .

b- If the ms is completely paralysed (cannot move to mid-line)

→. Ms transposition.

مثال لافتراض شلل في MR:



الحل: Collect some fibers from SR & some fibers from IR then pass throug ring to insert them instead of insertion of MR

3^{rd} nerve palsy Written مهم

1- Ptosis (due to lev. Paralysis).

2- Paralysis of EOM → deviation out (lat. R.). & no deppresion by SO (because in this position IO is resp.)

3- Paralysis of accommodation (paralysis of ciliary ms). Parasympathotic

4- No diplopia (due to ptosis).

5- Diltation of the pupil (paralysis of constrictor ms). → Dilated & irreactive

6- Proptosis (mild) due to loss of tone of the paralysed ms.

4^{th} nerve palsy: قراءة

1- Paralytic squint: the eye deviates up.

2- Diplopia: increase on looking down.

6th nerve palsy: if the rt. Side is affect قراءة

1- Rt. Conv. Squint (angle of squint is larger on looking to the rt).

2- Limitation of oc. Movement to the rt.

3- Dilopia: uncrossed and increase on looking to the rt.

4 -False p. on lokking to the rt.

5-Face turn to the rt.

Concomitant squint

Definition

It is a manifest squint which is not due to paralysis of EOM and in which the angle of squint is the same in all direction of gaze. (زاوية ثابتة مهما كان اتجاه النظر)

Aetiology

Binocular vision is fully developed between the ages of 6 months and 6 years. Any obstacle to binocular vision during this critical period would result in → ocular deviation.

High error	Low error
→ Accommodation	→ Tendency
→ +++ / ---	→ Imbalance
→ MR+++	Brain
→ MR---	Binocular vision (B.V. memory)

Such obstacles might be:

1- Refractive

(Uncorrected error of Refraction)

- In Hypermetropia (more than 3 D), the child accommodate to see cleary. Accommodation is associated with convegenceve→ Esotropia

- In Myopia, relaxation of accommodation& lack of convergence→ Exotropia (concomitant divergent squint)

2-Non-refractive

I-Congenital : esotropia is more common than exotropia

- Peripheral motor obtstacle: any anomaly of EOM .

anterior insertion
بدلاً من المكان الطبيعي

- Central obstacle (defective development of presumed fusion centre.

II- Sensory obstacle: due to monocular impaired vision.If the visual acuity in one eye is week,the brain will supress it→ unilateral squint

(no fusion)
ميعرفش B.V
لأن عمره ما اتعود
عينيه لأن هو
شايف بعين واحدة

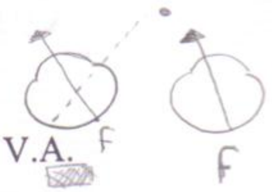
No movement in Covered test → Eccentric fixation
→ Paralytic squint
→ non-cooperative pt → Apparent squint
→ Non-seeing eye

Sequelae of concomitant squint:

With the onset of squint, the images of the object will fall on non-corresponding points of the retina a diplopia must occur. But, as the age of the patient is young, the brain finds a solution for this and diplopia doesn't persist. These solutions are:

Suppression:

- It is active neglect by the brain of the image seen by the squinting eye.
- It is temporary and occurs only when the non-squinting eye is fixing.
- If the fixing eye is covered → the squinitign eye will fix and with good V.A.



(sensory effort) لو أنا غطيت العين السليمة هيحاول brain يصلحها ويعدل مكانها وتشوف تاني لأن الدماغ أدرك أن هناك إلغاء للـ diplopia

Amblyopia ex-anopsia

- It is decrease in V.A in the squinting eye due to prolonged suppression.
- It occurs in unilateral squint. → not alternating (not as suppression)

Eccentric fixation:

- It occurs in some cases of amblyopia when the patient develop the ability to fix objects by a part of the retina other than the macula (false macula).
- It is due to dense suppression of the macula.

الدماغ اقتنع أن المكان اللي بيقع عليه الضوء في squint eye →

Investigation (diagnosis)

1- Test the ocular motility:

In the 6 cardinal directions → to exclude paralytic squint.

2- Measure the visual acuity:

- If equal in both eyes → alternating squint. (pt. Can fix by both eyes).
- Poor in the squiniting eye → unilateral squint (Rt. Or Lt).

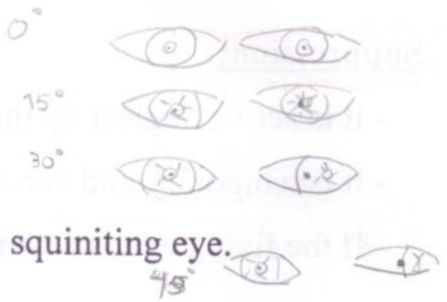
ECC, App sq → V. acuity
P. sq → ocular motility

3- The cover test:

- Ask the patient to fix an object (1st at a distance then at near).

- Cover the fixing eye:
  - The squiniting eye will fix.
  - The fixing eye will deviate.
- Remove the cover:
  - If the new position remains → alt. Sq.
  - If the original position retun → unilat. Sq.

4- Measurement of the angie of squint



ⓘ Corneal light reflex (Hirschberg test):

- Hold a tourch infront of the pt's nose.
- Observe the light reflex on the cornea of the squiniting eye.

(ii) Synoptophore (major amblyoscope)

- The synoptophore is an instrument, composed of 2 tubes which can be moved on a graded scale.
- Ask the pt. To look through the tubes.
- Then, ask him to rotate the tubes until the 2 test objects (at the tube end) are superimposed (e.g, the bird inside the cage).
- The angle between the 2 tubes = angle of squint.

آخر من كره
قيس من limbus
to point of
light

1m ~> 7-8°

(iii) Prism bar – cover test

Visuscope

→ Direct ophthalmoscope with + (cross) on its lens
→ Ask pt to fix (+) while exam his fundus
→ If ~> the + is seen on fovea = normal fixation
~> if seen outside fovea = eccentric fixation

5-Assessment of binocular vision

Binocular vision

Definition: it is the coordinated use of the 2 eyes to produce a single.

Values:
1- Steropsis (depth perception).
2- Larger field.
3- Optical defects and field defects (scotoma) in one eye are masked by the other.

Grades

1-Simultaneous macular perception (الدماغ ركب الصورتين على بعض بالعافية)

(the obility to perceive and superimpose the images of the 2 maculae).

2- Fusion (the ability to fuse 2 images with control ie., fusing 2 similar objects with missing detalis in each → one complete image).

3- Stereopsis (the ability to preceive the depth).

Assessment:

1- Synoptophore

Grade I: If the 2 test objects are superimposed.

Grade II: If 2 incomplete test object → one complete image.

Grade III: It is the ability to perceive the depth when viewing 2 slightly dissimilar objects.

2- Worth's 4 dot test:

Ask the patient to wear red-green goggles.

Then, ask him to look to the 4 colowed illuminated dots.

If the patient sees (possibilities ?).

if suppression → learn him B.V
if amblyopic → develop V.A then learn B.V

Treatment

Aim

- To build and restore binocular viision.
- To improve and preserve visual acuity of squinting eye.
- To improve the cosmetic appearance.

Time: as early as possible (before age of 7 ys).

- To avoid amblyopia.
- To allow binocular vision to develop.

Cases treated after age of 7 years are treated for cosmetic appearance.

Line of ttt

1. Glasses: correction of error of refraction,giving full cycloplegic refraction To relax accommodation (deduct 1.0 D for the working distance).
2. Amblyopia therapy: by covering the sound eye to improve the vision Of the amblyopic eye.

3-**Surgical ttt.**

Indication:

- No error.
- Residual angle (after glasses and orthoptic training).
- Age > 7 years (for cosmetic appearance).

Technique:

Weakening op.	Strengthening op.
1- Recession carrying the insertion backward	1- Resection shortens the ms → increase power.
2- Myotomy	2- Advancement carrying the insertion ant.

Rules

- Every 1mm (recession or resection) of M.R corrects 3°.
- Every 1mm (recession or resection) of L.R corrects 1.5°.
- If angle of squint is large → operate on several ms.
- In alternating squint, symmetrical surgery is required.

The orbit

Anatomy

The orbit is the socket of the eye

It is pyramidal in shape with the base anterior and the apex posterior.

Volume : 30 cm3. The globe ocuppies only ¼ of this volume.

Orbital fissures and formina Oral

1- Optic foramen (canal):

It transmits:
- Optic nerve.
- Ophthalmic artery.

أهم شئ

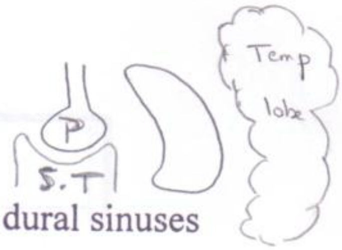
2- Superior orb. fissure

It transmits:
- 3,4,6 cranial nerves.
- The 3 branches of ophthalmic Nerve:
  - lacrimal
  - frontal
  - nasocil.
- Sympathetic root of ciliary gangliox.
- Ophthalmic vein.

3- Inferior orb. Fissure.

It transmits:
- Maxillary division of trigmental.
- Infra-orbital artery.

The cavernous sinus

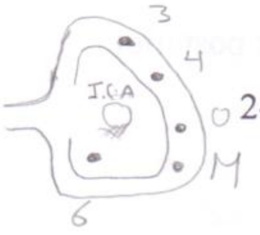


It is one of the paired dural sinuses

Relations : Lies lateral to sella turcica & medial to the Temporal lobe

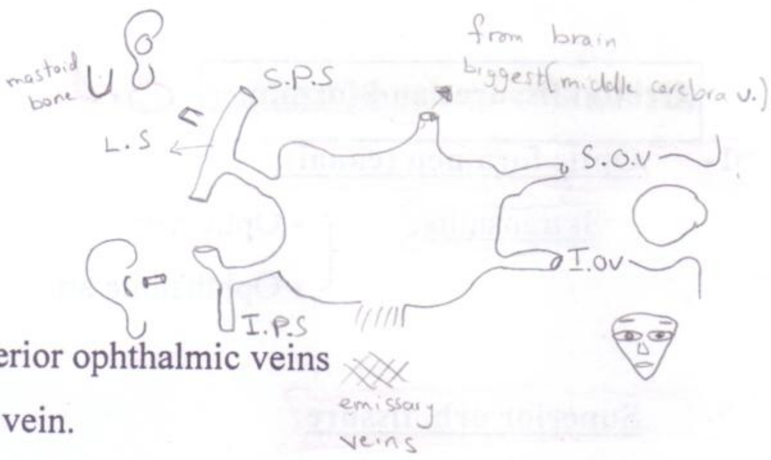
Contents: 1- The cavity contains:

- Internal carotid artery.
- Abducent nerve.



2- The lateral wall contains 9from above downward):

- 3rd n.
- 4th n.
- Ophth. n.
- Maxillary n.



Communication

1- **Anteriorly:** I) superior & inferior ophthalmic veins

II) central retinal vein.

(thus communicating with :

a- Eye & orbit.

b- Face: as oph. veins communicate with angular v.

2- **Posteriorly:** Superior & inferior petrosal sinuses

(thus communicating with :

a- Subcut. layer behind the ear:

- via the sup. Pet. sinus & mastoid emmissary veins

b- Middle ear (via inferior pet. Sinus & juglar vein

3- **Superiorly** : Middle cerebral vein

4- **Inferiorly**: emissary veins connecting it with Pterygoid plexus

(thus communicating with the mouth, pharynix & nasal sinuses)

5-**Medially**: Intercavernous sinuses to the other cav. Sinus.

Pseudoproptosis
→ Buphthalmus
→ high myopia
→ retracted lid

Diseases of the orbit

Proptosis = Exophthamus

Def: Passive protrusion of eye ball

Aetiology

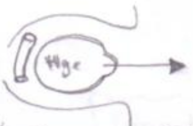
I- Congenital

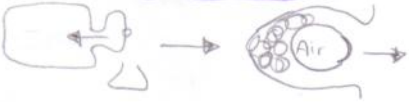
e.g, - Dermoid cyst.

- Meningo-encephalocele (defect in meninges → brain pushes eye forward

II- Acquired

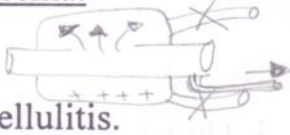
1- Traumatic e.g, Retrobular hematoma.





Surgical emphysema.

C.C-fistula
A-V shunt.



2- Inflammatory e.g. Acute: orbilal cellulitis.

Chronic: specific, T.B.

Non-specific: orbital pseudot tumor (releived by cortisol)

3- Neoplastic: (benign or malignant).

i- Primary: from orbital structures

- Lacrimal gl. Tumors (e.g, adenoma, mixed tumor).
- Oplic nerve tumors (e.g, glioma, meningioma).
- Haemangioma, fibroma, sarcoma, lymphomas. (Oral: Most common)
- Externsion from intra-ocular tumors (e.g, retinoblastoma)

ii- Secondary: from breast (F), bronchi (M). prostate

4- Endocrinal: Dythyroid Ophthalmopathy

5- Other causes:

a-Vascular: aneurisms, tumors, A.V shunts and varices.

b-Cyst: dermoid cyst, hydaltid cyst.

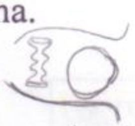
c-Paralysis of ex. Ocular ms (3rd n palsy). → fat will take upper hand due to loss of tone in muscles.

Diagnosis

مش مهم أوي

I- History:

- Trauma.
- Pain (indicates inflam. Or malignancy).
- Onset and course:
  - Ocute: inflam., trauma.
  - Intermittent: varices.
  - Slowly progressive, neoplastic.



II- Examination

A) General

1- ENT examination

2- Medical examination:

جراحة و باطنة

- Enalrged lymph nodes.
- Thyroid.
- Look for a primary.

B) Local

1- Inspection

i- Unilat or bilat: Unilat: other casues.

Bilateral:
- Reticulosis.
- Endocrinal.(Thyroid)
- Late cases of cav.s. thrombosis.

ii- Direction:
- Directly forward e.g, optic. N. tumors.
- Forward, down and in e.g, lac. Gl. tumors.
- Forward, down and out e.g, frontal mucocele.

Oral

Causes of Pulsating proptosis

iii- Pulsations e.g, in
- Oph. Artery. Aneumism.
- Vascular tumors (sarcoma).
- A.V shunt (carotid-cav.fistula).
- Meningo-encephalocele.

iv- Signs of inflammation
- Lid: oedema.
- Cong. Chemosis, hyperemia.

Cyst .. soft　Malignancy .. hard

2- Palpation: Consistency, fixation of the lesion. → Mobile: Cyst or granuloma → fixed: Malignancy

3- Auscultation: in carotid cavermous fistula → machingary murmer (Bruit)

4- Measurement of proptosis:

The distance between the:
- Lateral orb. Margin.
- Apex of the correa is measured.

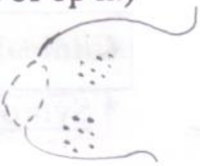
Normally → 15-17 mm.

The measurement is done by:
- i- Simple ruler.
- ii- Hertels exophthalmometer.

III- Investigations

A) Laboratory:
- Complete blood picture e.g, for leukemia.
- Tuberculin test.
- T3 , T4and TSH levels.
- Casoni test. → Hydatid cyst

B) Radiological 1- Plain x-ray
- Wide op. Foramen (e.g, glioma of op n.)
- Calcification (Maningioma).



2- Arteriography and venography (C.C fislula).

3- Ultrasonography.

4- C.T scan.

5- Magnetic resonance imaging (MRI).

C) Surgical:Biopsy → Excisional → Complete removal
→ Incisional → Take biopsy only.

IV- Treatment: Orbitotomy and excisional biopsy.

1- Treatment of the casue.

2- Orbitotomy: may be

A- Ant. Orbitotomy: for lesions in the ant. ½ of orbit.

B- lat. orbitotomy: for deeper lesions.

c-Trans-frontal orbitotomy: for orbital apex lesions. → break frontal bone → frontal lobe ع ـ → break frontal lobe again → to reach the tumor

Orbital celluitis

Written
= ١٩٩٥

Definition

It is acute suppurative inflammation of the orbial cellular tissue.

Causative organisms → Bacterial ⇝ Children: H. influenza
⇝ Adult: Staph & Strept
→ Fungus: Orbital mucormycosis (in immunocomp. pts)

Aetiology

سبب من الشارع **1- Direct infection**: by
- Penetrating wounds.
- Following operation (squnit. R.D.)

Oral

2- Spread of infection: from
- Sinuses. → Ethmoiditis
- Teeth.
- Globe (Endoplhalmitis).

Why common spread from ethmoiditis?
→ Due to thin wall of ethmoid bone.

سبب من الجسم **3- Blood borne**: as in septicemia

Clinical picture

* **Symptoms:**
- General: Fever, malaise.
- Local: Pain.
  Vision
  - Early: Good.
  - Late: decrease due to op. Neuritis

* **Signs**

① Oedema of hid and conj. → chemosis.

② Proptosis.

③ Limitation of oc. Motitity. → paralysed muscles
→ Pain of myositis

④ An abscess may form
& point through
- Lower fornix.
- Skin near lower orb. Margin.

} By gravity.

Complications

may Kill pt.

① Extension of infection: into
- Cranium → (br. Abscess, cav. Thrombosis).
- Op. Neuritis.
- Globe → endophthalmitis

② CRV thrombosis. ③- lagophthalmus → corneal ulcer ④ Bacteremia & Septicemia.

D.D.

See the table.

Treatment

1- **General** : antiobiotics

2- Local
- Hot fomentation.
- Incision: if abscess forms (deningage).

Cavernous sinus thrombosis

Written
هام جداً
(لا; كتاب
Anatomy of Cav. Sinus من هناك
حوض لعلم سيطب)

Definition

It is thrombophlembitis of the cav. Sinus

Aetiology

The infection may come from (dangerous triangle)

ⓐ- Face and orbit (ophthalmic vein).

ⓑ- Middle ear (inf. Petrosal sinus).

ⓒ- Mouth and pharynx (pterygoid plexus).

ⓓ- Blood borne (metastatic).

Clinical picture

As orbital cellulitis

* **Symptoms:**

- General: fever, malaise (marked).
- Local: Pain.
  Vision
  - Early: Good.
  - Late: decrease due to op. neuritis

  due to spread of inflammation through dura → to optic nerve

* **Signs:**

As orbital cellulitis

① Oedema: of lid and conj. → congestion not injection as in cellulitis
② Proptosis
③ Limitation of ocular motility.
④ Fundus engarged veins .
⑤ Oedema of mastoid region → diagnosed by pressure against skin over mastoid process

N.B. 1- Extension to the other sinus → bilat. Proptosis.
2- The earliest sign in the other eye → conv. Squint (6th n. paby).

Oral

أسرع طريقة لاكتشاف

→ bilateral contribution of bilateral sides.

Treatment:

The condition is very serious & coukld be fatal if not well treated

Prophylaxis: ttt of source of infection

Curative :

① **Massive antibiotics** ② **Anticoagulant**

Acute inflammatory proptosis?
2, 3 & 4

Written
V. imp

	1- **Endophthalmitis**	2- **Panophthalmitis**	3- **Orbital cellulitis**	4- **Cau. S. thrombosis**
Definition	It is suppurative inflam. Primarily in uveal tract (sclera is free) → presence of blood vessels.	It is severe sup. Inflam. Of uveal tract and other tissue including the outer coat	It is suppurative inflam of orbital cellular tissue	It is thrombophlebitis of the cav sinus
Aetiology	As infective iridocyclitis (see before)	As infective invidacyclitis (see before)	See (the orbit)	See (the orbit)
cl. picture				
symptome				
General.	Headache, fever	More (+++)	(+++)	Severe (++++)
Local				
Pain.	++	++	++	++
Vision	decrease to no P.L	no P.L.	good (early)	good (early)
Signs				
Lid.	Oedema	Oedema	Oedema	Oedema
Cong.	Chemosis+cil ing. due to iridocyclitis	Chemosis + cil inj by spread of infection	Chemesis + inj	Chemosis + conge
Cornea.	Hazy + kps. Keratic Percipitates	Hazy+ring absces	Clear	Clear.
Proptosis.	Absent	Present	Present	Present.
Oc. Motility.	Normal	Limited	Limited	Limited
Red reflex	Yellow (pus in vit.)	Yellow (Pus in vit)	Normal.	Normal
Treatment Antibiotics +	Seeing eye → vitrectomy non seeing → evisceration	Usually non secing → evisceration	Orbital abscess → drain	Anticoagulant + neurosurgical ttt

Oral

Meningo-encephalocele

قراءة

Herneation of the meninges and part of the brain through a defect in the orbital roof. It purlsnlea with the heart.

Surgical emphysema

قراءة

Air pases from the nasal sinuses into the orbit and subut tissue of the lids through a fracture of the ethmoidal bone: proplosis (increase blowing nase) and crepitation (are fett in lid).

In: 1- Presseure bondage. 2- Antibiotics 3- Don't blow the nose.

Arterior-venous fistula

قراءة

Cause: it is due to rupture of internal carotid artery as it passes in the cavernous sinus following severe trauma.

Treatment: 1- ligation of the common carotid a. in the neck.

2- ligation of the artery before and after the fistuta.

Enophthalmos

Written

يكتب بالتفصيل
كل من مكانه

1- Congenital. (microphthalmus)

2- Senite. by fat

3- Traumatic → Direct
→ fracture floor of orbit

4- After recovery from orb. Cellulitis (fibrosis).

most common ← 5- Horner's syndrome.

Operations of the orbit

1- Orbiotomy.

استئصال العين **2- Enucleation.**

- Principle: the eye ball is excised, while Conj., Op.n., Ex-oc.ms → are left. & then suture upper & lower Conjunctivae.

  (usually an antifical eye is inserted in place).

الأهم orally - Indications:

① To stop pain e.g, in absolute glaucoma.

② To save life e.g, in intraocular malignancy.

③ To save other eye e.g, in sympathetic ophthalmia. → to avoid spread of Pigmentation.

④ Xosmetic reasons e.g, in total ant. Staphyloma.

تفريغ العين **3-Evisceration**

- Principle: the cornea is excised and all the "contents" of the eye-ball are evacuated, while the sclera is left.

- Indications: endophthalmitis and panophthalmitis. To avoid infection to brain

sclera is intact

even if sclera infected → No enucleation to avoid spread of inf. to brain.

> **N.B.**
>
> Here enucleation cannot be done for fear of extension of infection along the sheath of op nerve → brain.

تفريغ الـ orbit **4-Orbital exentration:**

Principle: It is operation where all the contents of the orbit are removed inside the orbital periosteum. The lids may be also removed → all in one mas.

عن طريق قص Periosteum of orbit all around

Indications: ① Malignant orbital tumors.

② Malignant tumors of: lid., Cong ., Globe. (invading the orbit)

Provided that
- i- Periosteum is free.
- ii- No melastasis.

The pupil

Function

1- Regulates amount of light entering the eye.

2- Decrease aberration (chromatic) by cutting peripheral rays.

3- Increase depth of focus. The depth of focus is distance along which an object can be moved without becoming blurred for a known amount of accommodation. The smaller pupil. Greater depth of focus.

The light reflex

When light falls on eye:
- Ipsilateral pupil constricts (direct reflex).
- Also other pupil (concensual reflex).

***Pathway.**

1- **Stimulus:** light

2-**Receptors:** rods and cones.

3-**Afferent:**
1- Optic nerve.
2- Optic tract (temporal fibers).
3-Mid brain (pre-tectal nucleus) &from pretectal nucleus, a new neurone (intercalated neurone) delivers impulses to Edinger Westphal N.

4-**Centre:** edinger westphal nucleus (part of 3rd n. nucleus).

5-**Efferent:** 3rd n → ciliary ganglion (along nerve to I. Oblique as) → short ciliary nerves → constrictor pupillae ms.

N.B. The consensual reflex:

is due to crossing of fibres at

1- Chiasma (nasal fibres).

2- mid brain (intercalated neurones to 2 Edinger W.nucleus)

Stimulation of E. westphal nucleus

Pretectal n. (light reflex) ← → Frontal cortex (near reflex

7 : 30

The near reaction

When a near object is viewed 3 related (synkinesis) reflexes occur:

1- **Accommodation:** due to contraction of ciliary ms.

2- **Convergence:** due to contraction of 2 med. Recti.

3- **Miosis.**

Pathways:

1- Stimulus: blurring of image.

2- Receptors: rads and cones.

3- Afferent: Optic Nerve → Optic Tract.
→ Lat. Geniculate body → op. Radiation.
→ Occipital cortex → frontal cortex.
→ Internal capsule → Edinger w. nucleas.

4- Centre: edinger W. nucleus.b

5- Efferent: as the light reflex.

lesion in intercalated fibers:
→ No miosis
→ There is near reflex

Argyl Robertson pupil

(light-near dissociation)

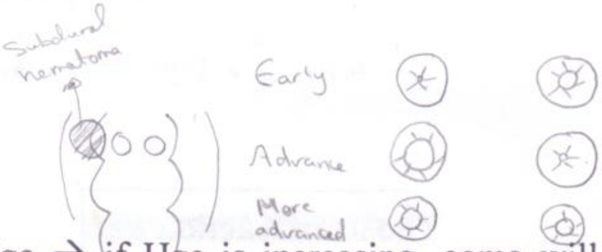
1- It is small pupil (usually bilateral) → due to continous irritation of nucleus by the lesion

2- That doesnit react to light but reacts to accom

3- It dilates poorly in dark. → due to incomplete relaxation by stimulated sph. pupillae

*Site of lesion: intercalated neurone.

*Cause:
- Commonest: neurosyphilis (tabes & GPI).
- Other causes: D.M, encephalitis, trauma.



Hutchinson's pupil

In subdural Hge → if Hge is increasing, coma will deepen and pupils show the following changes

	Ipsilateral pupil	Contralateral pupil
Early stage	Constricted	RRR
Advanced (As by Atropine)	Dilated and irreactive	Constricted by irritation
More advanced (Death)	Dilated irreactive	Dilated and irreactive

A dilated pupil + increased coma → is an indication for cerebral decomperession

Marcus- Gunn Pupil Def: Parddoxical effect

Cause: Unilateral or bilateral assymetrical op. n. lesion

Characters: Direct reflex : affected Consensual reflex : normal

Oral Diagnosed by : Swinging flash light test.

Horner Syndrome: (oculo-sympathetic palsy)

Cause: lesions of sympathetic innervation of the eye, due to

- brain stem : vascular or demylinating diseases.
- Bronchogenic carcinoma or nasopharyngeal tumors

Characters :

1- Ptosis (due to weakness of Muller ms.)

2-Miosis (unopposed action of sphincter pupillae)

3- Anhydrosis (reduced sweating)

4- Enophthalmos

Afferent pupillary defect

→ Lesion in optic nerve

E.g:

1 CRAO

2 Optic nerve atrophy

(Oral هام)

Causes of miosis and mydriasis

	Miosis	Mydriasis
1- Physiological	Light and near reflexes During sleep (parasym.) 3rd stage of anaesthesia old age (by sclerosis of sph. pupillae) -newly born (Due to delayed development of dilator)	Withdrwal of light (Dark) Emotion (sympath) 2nd stage by high adrenaline
2- Drugs	Miotics (pilocarpine) Morphine (opium) poisoning	Mydriatics Datura poisoning
3- Local diseases	Trauma Iridocyclitis by irritation of sph. pup. Hypermetropia (عين صغيرة أصلاً) Hypotony	Trauma Acute cong. Glaucoma. Myopia. (عين كبيرة أصلاً) Blind eye: CRAO, Op. Atrophy (زي ما يكون ماشي في الضلمة)
4- Neurological	Horner's syndrome Argyl Robertson pupil Pontine hge (by mechanical pressure on nuclei) Irritative stage of cerebral compression (hutchinson)	→ 3rd n. palsy → coma except in (Oral هام) 1- morphine poisoning 2- pontine Hge → - paralytic stage of Hut. Pupil. → - 4th stage of anaesthesia

Neuro-ophthalmology

Anatomy

1-otoreceptors : Rods & cones

which synapses with bipolar cells → ganglion cells

2-Axons of ganglion cells run in the n.f.layer of retina & converge to

To form the **Optic nerve**.

3- At the **Optic chiasma** : the nasal fibers decussate to reach the **Op.tract** On the opposite side,while the temporal fibers pass uncrossed to the Ipsilateral tract. Finally the fibers reach the LGB where they synapse.

4-From **the LGB** , new axons spread out over broad area of temporal&

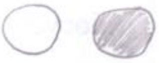
Parietal lobes to formthe optic radiation.

5-Fibers in **Op. radiation** end in the visual cortex, which includes:

- Area 17 (1ry visual area-striate cortex.
- Areas 18 & 19 (visual association areas)

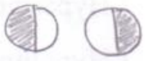
I- Visual pathway lesions:

1- Op. nerve Lesions: ipsilateral blindness.



(oral الـ ١٨ هم

2- Op. chiasma lesions: bitemporal hemianopia.



مش شايف الناحيتين العكسية

3- Op. tract lrsions : contralat. Homo. Hemianopia (+ loss of light reflex)

4- Lat. Gen. body : contralat. Homo. hemianopia (+ preserved light reflex)

5- op. radiation

Upper fibres (parietal lobe) → Contr. Homo.inferior quadrantic defect

lower fibres (temporal lobe) → Cont. Homo.superior quadrantic defect

6- Occipital cortex

→ contranlat. Homo. hemianopia with macular sparing

Due to i-Large area of representation.

ii-Bilat. Representation.

iii-Double blood supply. → Posterior & middle cerebral arteries. ١٢ vc. ٤٢٥١

II- Ocular manifestation of brain tumors

Oral ضعيف

A) General signs (of increase ICT).

[illegible] 1- Triad of : headache, projactile vomiting, bluring of vision.

2- False localizing signs: e.g, 6th n. palsy (squint & diplopia)

(being: slender and long course against bony prominances.

B) Focal signs:

Which help in localization

1- Frontal lobe: → Foster-kennedy syndrome.

2- Parietal lobe.: → contra lat. Inferior quadrantic defect.

3- Temporal lobe:
- Contra lat superior quadrantic defect
- Visual halucination.

4- Occipital lobe:
- Contra lat. Homo. Hemianopia with mac. Sparing.
- Visual halucination.

5- Cerebellum: - Nystagmus (increased on looking to the side of lesion)

6- Pituitary - Bitemporal hemianopia + endocrinal changes.

Important symptoms

I- Visual disturbances

A) Diminution of vision:

i- Sudden loss of vision

1- CRAO. 2- Amaurosis fugax.

3- Hysteria. 4- Rupture globe.

ii- Rapid decrease of vision:

a- In few hours

1- CRVO 2- Acute congestive glaucoma.

3- Commotio R. 4- Uraemic amaurosis.

5- Vitreous hge.

b- In few days

1- Keratitis, 2- iritis, choroiditis

3- R.D 4- Optic neuritis.

iii- Gradual decrease vision

1- Senile cataract. 2- Glaucoma simplex.

3- 1ry op. Atrophy. 4- Degenerative myopia.

5- Retinitis pigmentosa 6- Senide macular deg.

7- Retinopathy

B) The opsias

1- Metamorphopsia: objects appear distorted.

2- Macropsia: objects appear large.

3- Micropsia: objects appear smaller.

* Cause: distortion of the "macular" rods and cones as in:

1-Retinitis. 2-Choroiditis. 3-RD 4-Commotio Retinae.

4- Photopsia: seeing flashes of light.

* Cause: stimulation of rods and cones as in:

1- Retinitis. 2-Choroiditis. 3-RD. 4-High myopia.

5- Chromatopsia: coloured vision:

i- Erythropsia (red vision): occurs after cataract extraction.

ii- Xanthopsia (yellow vision): occurs in jundice.

iii- Cyanopsia (blue vision): occurs in hysteria.

C) Night blindness

-Definition: it means difficulty to see in dim illumination.

- Causes: 1- General: - Vit A deficiency. - Liver diseases& alcoholics.
  2- Local: - Congenital. - Hysterical.
  - Peripheral lens opacities. - High myopia. (malignant)
  - R. pigmentosa. - Siderosis bulbi.
  - OAG. - Quinine amblyopia

D) Day blindness

- Definition: it is difficulty to see in bright illumination.
- Causes: 1- Central corneal opacity.
  2- Central lens opacity (macular).
  3- Central retinal lesion (macular degeneration).

E) Colour blindness

* Causes: 1- Congenital: most, common type.
2- Acquierd: as in papilloedema and papillitis.

* Tests: i- Isochromatic charts (Ishihara) ii- Coloured beads.

F)Photophobia:

1- Cong gl. 2- Coneal abrasion 3- FB (corneal or conjunctival)

4- Iridocyclitis 5- Acute congestive glaucoma

G) Diplopia (double vision)

* Types 1- Binocular diplopia: ie,disappears if either eye is covered

e.g, in.

i- Physiological ii- Paralytic sq.

iii-Anisometropia or unilat. aphakia: corrected with glasses

(هام جداً) 2- Uniocular diplopia

Present even if the normal eye is covered e.g, in

i- Incipient cat. ii-Subluxation.

iii- Iridodialysis. Iv- Irregular astigmatsim

II- Ocular pain, discomfort, Headache

1-Mild Discomfort: as in i-conjunctivitis. ii- Eye strain.

2-Itching pain: as in i- Allergic conjuctivitis(especially vernal) (Spring catarrh)

ii- Blepharitis (esp angular)

3-Severe pain: as in i-corneal ulcer (stitching), FB.

ii- iritis (neuralgic),

iii- acute congestive gl. (bursting).

4- Orbital Pain :

i- Retrobulbar neuritis

ii- Orbital cellulitis

iii- eye strain (> in hyperopia)

iv- orbital trauma

v- Myositis

vi- Obital periostitis

Headache: found, with hypertension, increae ICT.

Staphyloma

Written

Definition

لازم تقولها

it is an ectasia of the outer coat of the eye (in which the uvea is sharing)

Aetiology:

1- Corneal disease e.g, perforated cor. Ulcer.

sclera ضعيفة 2- Scleral diseases e.g, high axial myopia.

ضغط عالي 3. Incresae IOP e.g, absolute glaucoma:

Types

1.Corneal (anterior staphyloma)

Aetiology: Large corenal perforation → irirs prolapse

The weak scar (large defet) cannot withstand the IOP

(due to closure of the angle by the iris) → ectasia

D.D.: From keratectasia (see the cornea)

ttt: see the cornea

2- Scleral

Types: تحدث في أضعف الأماكن في جدار العين

(between 2 areas)

at limbus ← (1) Intercalary: lined by the root of iris. → Canal of Schlemm

(2) Ciliary: lined by ciliary body. → art. vessels & nerves

at equator (خط الاستوا بتاع العين) ← (3) Equatorial: lined by the choroid. → in vortex veins

(1), (2), (3) result from glaucoma, scleritis

(4) Posterior staphyloma: at the posterior pole,

- occurs "only in high myopia,
- seen by ophthalmoscope, or U.S

Sclera

Anatomy:

It is the posterior 5/6 of the outer coat.

It is formed of : 1- Tenon's capsule (كيس حول العين زي الـ synovial sheath لسهولة حركة العين) 2- Episclera = collagen between sclera & tenon's capsule. 3- Lamina fusca

Perforated by:

(1) Arteries: ant, ciliary a., & post. Ciliary a. (2 long & 6 short)

(2) Veins: ant. Ciliary V. & vortex veins. دوامة

(3) Nerves: short (6-10) & long ciliary (2) nerves. [illegible]

Inflammation

Superficial→ episcleritis. Deep → scleritis

Aetiology: (1) Part of a collagen disease e.g, Rheumatiod arthritis, gout.

الأكل، الشيكولاتة، والدوا (2) Allergy to endogenous toxins.

Cl picture: It may be diffuse or nodular inflammation. → due to antigen-antibody reaction (allergy)

inflammation

D.D.: Nodular episcleritis from phylcten.

Treatment: (1) ttt of the cause (2) Cortico steroids. → أدي علاج كويس
→ anti inflammatory
→ anti allergic

Blue sclera

Oral & Slide

بياض العين [illegible] sclera

Cause: **(1) Physiological :** as in infants & children

(2)Pathological : (congenital weak outer coat)

1. Buphthalmos = congenital glaucoma 2. High myopia 3. Over a staphyloma (الأزرق في مكان محدد not diffuse)

4. Osteogenesis imperfecta: blue sclera,deafness & fragile bone

In all these cases, the sclera is thin showing the underlying uvea (superficial layer [illegible] choroidal veins [illegible])